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HEXYLRESORCINOL AS AN ANTHELMINTIC

ITS EFFICIENCY AGAINST THE INTESTINAL PARASITES OF MAN

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Many of the anthelmintics that have been shown by means of critical tests to possess a high degree of efficiency are poisonous substances, for which reason their use has been followed in a number of instances by symptoms of intoxication and sometimes death. According to Lamson and Ward (1932) the only anthelmintics that from a practical standpoint can be considered as both safe and efficient are tetrachlorethylene and hexylresorcinol. The more recent observations of Christensen and Lynch (1933), however, on the effect of these two drugs on the host, have shown that the former, even in single therapeutic doses, depresses the heart and the respiration and produces pathologic changes in the small intestine, liver, and heart. In the case of hexylresorcinol no apparent symptomatic disturbances and only slight changes in the liver and heart tissues were noted.

Hexylresorcinol had long been used as a genito-urinary disinfectant before it was introduced as an ascaricide by Lamson, Ward, and Brown in 1930. It has since then been tried in different parts of the world and found to be effective, not only against ascarids but also against hookworms and other intestinal parasites. So far as we are aware it has not yet been tried extensively in the Philippines, for which reason it was decided to test its efficacy under local field conditions.

MATERIALS AND METHODS

The hexylresorcinol used was obtained directly from Messrs. Sharp and Dohme through the courtesy of their Philippine representative, Mr. C. S. Lounsbury. It was received in two shipments: the first shipment was in the form of gelatin capsules each containing 0.166 gram and the second shipment in the form of chocolate-colored sugar-coated pills each containing 0.2 gram of hexylresorcinol. We agree with the other workers who have used the drug as to the impracticability of gelatin capsules in a warm and moist environment such as that met with in the Philippines. Due most probably to the reaction of the hexylresorcinol with the gelatin, which reaction was accelerated by the conditions of the climate, the majority of the capsules arrived broken and therefore unfit for use. Those that were still intact were soft and broke easily during the process of swallowing. Besides, as will be shown later, the reaction of the drug with the gelatin seemed to have rendered it less effective as an anthelmintic.

The treatments were administered under field conditions, each individual receiving only a single treatment. The drug was given on an empty stomach early in the morning. The patients were allowed to go about their daily activities but were advised to abstain from food for at least four hours after taking the medicine. In some cases a saline cathartic (sodium sulphate) was given twenty-four hours after the anthelmintic.

The following doses recommended by the manufacturers were given:

To adults and children over 12 years of age 5 pills or 6 capsules.
To children 8 to 12 years of age 4 pills or 5 capsules.
To children 6 to 8 years of age 3 pills or 4 capsules.
To children under 6 years of age 2 pills or 3 capsules.

The efficacy of the drug was judged from the results of the examination of fæcal samples by the Stoll technic (Stoll and Hausheer, 1926) before and ten to fourteen days after the treatment. A total of 861 persons, representing both sexes and all ages from 4 years up, were treated, but only 448 of these submitted specimens for reëxamination.

EFFECT OF HEXYLRESORCINOL ON THE HOST

Of the 861 persons treated not one showed any noteworthy symptom of intoxication. A few individuals complained of slight symptoms of either gastric or intestinal irritation accompanied by headache and dizziness, but these disappeared rapidly without any treatment and in many cases they were probably due more to the fasting than to the effect of the drug. One woman, who vomited after swallowing the pills, ascribed it to the medicine; but it turned out that she was a very nervous individual and an ex-inmate of an insane asylum.

Lamson and Ward (1931) have called attention to the local irritating action of the drug on the mucous membranes of the mouth and tongue if the capsules or pills break in the mouth or are chewed against advice. When this happens the mucosa may become blanched and the superficial epithelium may even peel off. Several of our cases, most of whom were young children, met with such accidents, but they did not materially suffer from the lesions. Only the woman referred to above, who vomited the drug through the nose, was inconvenienced for several days as the result of severe burns in the nasal passages.

EFFICIENCY OF HEXYLRESORCINOL AGAINST DIFFERENT PARASITES

The observations recorded in this paper were conducted for the purpose of ascertaining the efficiency of hexylresorcinol against *Ascaris lumbricoides, Trichuris trichiura,* hookworms,[1] and other intestinal worms infesting man. The results obtained do not appear to be as satisfactory as they should be due to the poor coöperation obtained from several of the individuals treated. These partook of food soon after taking the anthelmintic, as a consequence of which they showed very little, if any, diminution in their egg counts after the treatment. These cases have been included in the tabulation of the data in order to determine, as aptly expressed by Molloy (1933), "the results which might be obtained by administering the drug under the customary field conditions."

TABLE 1.—*Ascaris: Efficiency of hexylresorcinol in gelatin capsules, followed in twenty-four hours by a purgative of sodium sulphate. (Pardo, Cebu, Cebu.)*

Cases	88
Egg count per cc (formed basis):	
Before treatment	2,420,500
After treatment	1,216,100
Reduction, per cent	49.76
Negative after treatment, per cent	11.36

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[1] It has been shown by several investigators that the prevailing species of hookworm in native Filipinos is *Necator americanus.*

TABLE 2.—*Ascaris: Efficiency of hexylresorcinol pills, not followed by a purgative.* (*Manila.*)

Cases	232
Egg count per cc (formed basis):	
Before treatment	16,991,000
After treatment	3,051,800
Reduction, per cent	82.04
Negative after treatment, per cent	53.45

TABLE 3.—*Ascaris: Efficiency of hexylresorcinol pills, followed in twenty-four hours by a purgative of sodium sulphate.* (*Baao, Camarines Sur, and Cabuyao, Laguna.*)

Cases	61
Egg count per cc (formed basis):	
Before treatment	2,237,400
After treatment	333,800
Reduction, per cent	85.08
Negative after treatment, per cent	63.93

Effect on ascaris.—The effect of hexylresorcinol on ascaris was observed in a total of 381 cases, which, as shown in Tables 1, 2, and 3, were divided into three groups. Table 1 includes individuals who received the anthelmintic in gelatin capsules (followed by a purgative), Table 2 those who were given pills but were not purged, and Table 3 those who received pills, followed in twenty-four hours by a purgative of sodium sulphate. It will be seen from these tables that the effect of the capsules was much less than that of the pills. With the capsules the average reduction in the egg count was only about 50 per cent and but 11 per cent of those treated, a large number of whom were lightly infested cases with egg counts of less than 3,000 per cc, were cleared entirely of their ascaris burden. When pills were used the reduction in the egg count was 82 to 85 per cent and the number of those found negative after treatment was larger (53 to 64 per cent) and included several heavily infested cases with counts up to 150,000 eggs per cc. In the majority of instances, however, the average efficiency of the drug in heavy ascaris infestations was noted to be about 80 per cent. As observed by Molloy (1933), the anthelmintic was slightly more effective when followed by a saline purgative twenty-four hours after (Table 3).

Effect on hookworms.—The results of the treatments against hookworms are summarized in Tables 4 and 5, in which it is shown that the gelatin capsules compared with the pills were no more efficient against these parasites than against ascaris.

In the series in which gelatin capsules were used (Table 4) the reduction in the egg count was only 33.7 per cent and the percentage of those found negative after the treatment 7.7. In the group in which pills were given (Table 5) there was a reduction of 74 per cent and 25.4 per cent of the cases were found negative after the treatment. We believe that these latter figures, which agree quite closely with those obtained by Molloy in Central America, represent more or less accurately the efficiency of a single dose of hexylresorcinol against human hookworms under field conditions.

TABLE 4.—*Hookworms: Efficiency of hexylresorcinol in gelatin capsules, followed in twenty-four hours by sodium sulphate. (Pardo, Cebu, Cebu.)*

Cases	103
Egg count per cc (formed basis):	
Before treatment	524,200
After treatment	347,600
Reduction, per cent	33.69
Negative after treatment, per cent	7.76

TABLE 5.—*Hookworms: Efficiency of hexylresorcinol pills, followed in twenty-four hours by a purgative of sodium sulphate. (Baao, Camarines Sur, and Cabuyao, Laguna.)*

Cases	62
Egg count per cc (formed basis):	
Before treatment	542,000
After treatment	140,200
Reduction, per cent	74.13
Negative after treatment, per cent	25.40

TABLE 6.—*Trichuris: Efficiency of hexylresorcinol in gelatin capsules, followed in twenty-four hours by sodium sulphate. (Pardo, Cebu, Cebu.)*

Cases	99
Egg count per cc (formed basis):	
Before treatment	566,800
After treatment	404,600
Reduction, per cent	28.62
Negative after treatment, per cent	5.05

TABLE 7.—*Trichuris: Efficiency of hexylresorcinol pills, not followed by a purgative. (Manila.)*

Cases	229
Egg count per cc (formed basis):	
Before treatment	1,539,000
After treatment	1,109,200
Reduction, per cent	27.93
Negative after treatment, per cent	8.73

TABLE 8.—*Trichuris: Efficiency of hexylresorcinol pills, followed in twenty-four hours by sodium sulphate. (Baao, Camarines Sur, and Cabuyao, Laguna.)*

Cases	61
Egg count per cc (formed basis):	
Before treatment	184,200
After treatment	91,200
Reduction, per cent	50.5
Negative after treatment, per cent	18.0

Effect against trichuris.—The results of the treatments against trichuris are summarized in Tables 6, 7, and 8. In the series in which the anthelmintic was given in gelatin capsules (Table 6) a reduction of almost 29 per cent in the egg count was noted and 5 per cent of the cases were negative after the treatment. In the series in which pills were administered but not followed by a purgative (Table 7), the percentages of egg reduction and apparently complete cures were 27.9 and 8.7, respectively. In the last series in which the pills were followed by sodium sulphate after twenty-four hours (Table 8), the results were 50.5 per cent for egg-count reduction and 18 per cent for the number of cases found negative after the treatment.

TABLE 9.—*Unusual trichuris egg counts before and after treatment with hexylresorcinol.*

Case No.	Egg count per cc (formed basis).		Case No.	Egg count per cc (formed basis).	
	Before treatment.	After treatment.		Before treatment.	After treatment.
72	2,800	14,400	602	52,600	92,000
116	800	3,600	616	3,000	18,000
217	2,800	6,400	623	2,000	13,000
218	1,600	4,400	652	8,800	20,200
271	2,000	5,600	640	400	4,800
390	200	2,400	682	600	4,400
392	8,800	19,800	686	3,400	18,600
536	200	4,600	689	1,400	6,800
448	3,200	7,200	690	2,400	9,600
594	3,200	8,000			

According to the above figures, the hexylresorcinol pills, if not followed by a purgative, were not superior to the gelatin capsules; but if followed by a purgative their efficiency against trichuris was almost doubled. Too much importance, however, must not be attached to these results, for, as noted by Hall (1929) and Molloy (1933), differences in trichuris egg counts before and after treatments cannot always be relied upon as an accurate index of the efficacy of an anthelmintic. Both of these investigators met with cases whose egg counts were increased after treatment in spite of the fact that female worms were expelled by some of them (Hall's cases). In our series there were forty-six such cases, in some of which, as shown in Table 9, the increases registered after treatment were so great that the differences could not have been due to technical errors. Molloy would explain these discrepancies as due to the irregular and easily upset egg laying habits of whipworms; but, according to Hall, "there is some likelihood that the contents of the cecum, where whipworm eggs are deposited, may be expelled more or less spasmodically and intermittently, and that an egg count for any given sample of feces might be a variable."

Effect on Enterobius vermicularis and Tænia saginata.—Although our observations on the effect of hexylresorcinol on these two parasites are too limited to be of much significance, we have decided to mention them briefly in order to complete our report. In most of the cases the anthelmintic was administered orally in pill form followed in twenty-four hours by sodium sulphate. In some of the enterobius cases instead of the purgative high enemata of a 1 : 1,000 solution of hexylresorcinol in water were given.

Enterobius vermicularis.—There were 17 persons who were found by microscopical examination to harbor this parasite. In view of the inability to make differential egg counts before and after treatment in pinworm infestations as practiced in the case of ascaris, trichuris, and hookworms, the determination of the efficiency of the drug consisted in finding out if due to the treatment dead worms were expelled and if the patients were relieved from the well-known symptoms of enterobius infestation. Based on these considerations our results were satisfactory in 13 out of the 17 cases treated. In two cases who were given hexylresorcinol enemata, the number of pinworms collected in the fæcal screenings of each individual was so great that there is no doubt as to the lethal effect of the drug on the parasite.

Tænia saginata.—Only two cases were found to harbor this tapeworm. In contrast to the observations reported by Lamson and Ward (1932), neither of these two cases was permanently cured, indicating that the drug was unable to cause the expulsion of the head of the parasite.

SUMMARY

Observations were made on the efficiency of hexylresorcinol against different types of human intestinal worms under field conditions in the Philippines.

The drug was given in hard gelatin capsules and in the form of sugar-coated pills, adopting the doses recommended by the manufacturers. It was administered early in the morning on an empty stomach and the patients were advised not to take food for at least four hours afterwards. Each patient received only a single treatment.

A total of 861 individuals, representing both sexes and all ages from 4 years up, were treated. No important toxic symptoms from the drug were observed. A few cases complained of slight symptoms of either gastric or intestinal irritation but these quickly disappeared without any treatment. Some presented burns in their mouths due to the breaking of gelatin capsules, but they did not materially suffer from the lesions. One woman, however, who vomited through her nostrils soon after taking the drug, suffered much inconvenience due to severe burns in the nasal passages.

Hexylresorcinol was found to suffer in anthelmintic efficiency when placed in gelatin capsules, due most probably to the reaction of the drug with the gelatin. The capsules themselves undergo rapid deterioration, especially in a hot and moist climate, as a result of which they either become unfit for use or, if they remain intact, they are easily broken in the mouth during the process of swallowing.

The sugar-coated pills, besides being more efficacious than the gelatin capsules, did not appear to be affected by climatic conditions. Their anthelmintic efficiency was appreciably increased by a saline purgative twenty-four hours after their administration.

In infestations with ascaris and hookworms the administration of single doses of hexylresorcinol pills removed from 82 to 85 per cent of the former parasite and 74 per cent of the latter. Of the ascaris cases 53 to 64 per cent were found negative after the treatment and of the hookworm cases 25.4 per cent.

In trichuris infestations hexylresorcinol was found to be apparently effective, but some doubt is expressed as to the accuracy of judging the efficiency of the drug from the results of differential egg counts before and after treatment.

Observations on a limited number of cases showed that hexylresorcinol is also effective against the human pinworm (*Enterobius vermicularis*) but not against the tapeworm (*Tænia saginata*).

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AVIAN MALARIA STUDIES, IX

ATABRINE AS A PROPHYLACTIC DRUG IN SPOROZOITE INFECTIONS OF AVIAN MALARIA[1]

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INTRODUCTION

Atabrine is a recently marketed synthetic antimalaria drug which is said to be a substituted alkylaminoalkylamino acridine derivative. It was originally called erion and as sold in some countries its name is spelled atebrin. It is a yellow powder, soluble in water. The aqueous solution has a neutral reaction. Its chemical constitution has not yet been published.

In the Philippines atabrine is obtainable by the public only in tablets of 0.1 gram each and the usual dose for malaria in an adult is 15 tablets, taken at the rate of one, three times a day. For the purposes of this experiment it was possible to obtain, through the courtesy of the Winthrop Chemical Company, ampules containing exactly 0.2 gram of atabrine powder, soluble in water.

These experiments were undertaken to test the value of atabrine as a true or "casual" prophylactic in sporozoite infections of avian malaria. That is to say, an attempt was made to find out if atabrine has a lethal or inhibitory effect on sporozoites. Avian malaria was used because it was not feasible to work locally either with human or simian malaria under existing conditions. There is no strain of simian malaria available in the Philippines and there are no facilities for malaria experimentation on humans.

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[1] These studies are from the laboratory of Malaria Investigations of which the author is chief and which is jointly supported by the Bureau of Science, Manila, and the International Health Division of the Rockefeller Foundation. Mr. Andres M. Nono, Misses Capistrano and Villacorta, and Mrs. Ramos, all of the staff of Malaria Investigations, assisted at various times during the experiments. I am indebted to the Winthrop Chemical Company, Dr. G. Schwab, local manager, for the atabrine used in these experiments.

But a review of experiments in avian malaria reveals that results have never been misleading as regards human malaria. On the contrary excellent leads have come from avian studies ever since Ross, by using *Culex* mosquitoes and birds, demonstrated that malaria is a mosquito-borne disease. In the development of the synthetic drugs plasmochin and atabrine, for instance, avian malaria has been of the greatest service and the results observed in birds have invariably been significant when referred to human malaria. So, too, in testing plasmochin and quinine as prophylactics against malaria the results in birds have indicated what could be reasonably expected in humans.

For example, it was at first believed that plasmochin was a true prophylactic in human malaria, and James(1, 2) reported some human experiments which seemed to show this. But the studies of Russell and Nono(3) and of Tate and Vincent(4) showed very clearly that plasmochin will not effectively destroy or inhibit the sporozoites of avian malaria. This proved to be the case in human malaria for the cases originally believed to have been protected were found actually to have been infected. The effect of the drug had been to prevent immediate symptoms but not to prevent infection. (See James,(5, 6) Swellengrebel and de Buck,(7, 8) Russell and Holt.(9)) Here was a case where the first human experiments were definitely misleading, but the avian experiments pointed to the true situation.

ATABRINE AS A PROPHYLACTIC IN HUMAN MALARIA

There are few reports regarding the prophylactic action of atabrine in human malaria. Napier and das Gupta(10) reported one case in which atabrine seemed to protect a volunteer from mosquito-borne malaria. The treatment was begun the day before infection and was continued for five days thereafter. This case was negative for at least two months although controls became positive. The effect of atabrine here was more likely therapeutic than strictly prophylactic.

Soesilo(11, 12) reported that six among twenty-one volunteers, treated prophylactically with atabrine and bitten by infected mosquitoes, showed parasites in their blood after twelve to twenty-four days. The other fifteen remained negative for at least seven months. Ten of eleven volunteers, used as controls and not given atabrine, showed parasites in their blood after eight to thirteen days. Soesilo gave total maximum prophylactic doses of 1.4 grams and minimum of 0.6 gram. In the cases that became positive in spite of the atabrine the prepatent period was prolonged.

James(13) reported some experiments at the Devon Mental Hospital which may be summarized as follows:

A summary of James's experiments.

Group.	Case No.	Bites by infected mosquitoes (*A. maculipennis*).	Result (up to 30th day).	
			Attack.	Length of prepatent period.
				Days.
No drug	1	8	Yes	12
	2	6	Yes	10
Quinine gr. XV daily for 6 days	3	5	Yes	11
	4	4	Yes	11
	5	5	Yes	10
	6	8	Yes	12
	7	6	Yes	14
Phenoquinine 6 tablets daily for 6 days	8	5	Yes	10
	9	8	Yes	11
	10	3	Yes	11
	11	5	Yes	10
	12	5	Yes	11
Atabrine (atebrin) 3 tablets daily for 6 days	13	7	No	No parasites seen up to 30 days after infection.
	14	7	No	
	15	3	No	
	16	5	No	
	17	5	No	

In these experiments the first dose of atabrine was given four hours before the mosquitoes were allowed to bite the patient. The results indicate that atabrine certainly postponed the attacks and may have entirely prevented them. But the effect probably was therapeutic as the doses continued for five days after infection. There was no evidence in the experiments showing any action of atabrine on the sporozoites of human malaria. Yet these reports from Soesilo(12) and James(13) indicate that in a practical way atabrine may have prophylactic value.

GENERAL PROCEDURE

It is not necessary to describe the general procedure of these experiments because it has been fully discussed in previous papers.(3, 14, 15) The same strains of *Culex fatigans* (*quinquefasciatus*) and of *Plasmodium capistrani* were used, and the same technic was followed.

RESULTS OF EXPERIMENTS

Seven experiments, on sixty-five canaries, are described in this report.

EXPERIMENT I

Used twenty birds, T34 to T53. Birds T36, 37, 38, 39, and 40, each received an intramuscular injection of 0.0005 gram atabrine in 0.1 cc distilled water into pectoral muscles on February 7, 8, and 9 at 3 p. m. On February 9 at 10.30 a. m. birds T34 to T53 each received into the pectoral muscles 0.15 cc of a mixture of normal saline and macerated thorax tissue from infected mosquitoes. Thorax tissue from a total of 120 mosquitoes in 4 cc of saline had been macerated in a small mortar and pestle. Each bird therefore received approximately the equivalent of the thorax contents of 4.5 mosquitoes. An examination of this mixture after all injections had been given revealed numerous viable sporozoites. A total of twenty-eight mosquitoes from this lot had previously been dissected and 32 per cent of the salivary glands contained sporozoites.

Of the twenty birds used in this experiment fifteen died very promptly after receiving the sporozoite injection, as follows:

February 10: Birds T36, 45, 48, 50, 51, and 52.

February 11: Birds T35, 42, 43, 44, 46, 47, and 49.

February 13: Bird T40.

Birds T34, 37, 38, 41, and 53 were still alive March 22, and each of them became infected with malaria.

Bird No.	Blood-smear examinations.	
	Negative.	Positive.
T34	January 23; February 12, 13, 14, 15, 16; March 4.	February 17, 18, 19, 20, 22, 25, 26, 28; March 2.
T37	January 23; February 12, 13, 14; March 2, 4.	February 14, 16, 17, 18, 20, 22, 25, 26, 28.
T38	January 23; February 12, 13, 14, 15, 16; March 2, 4.	February 17, 18, 19, 20, 22, 25, 26, 28.
T41	February 3, 12, 13, 14, 15, 16, 17, 18	February 19, 20, 21, 23, 24, 25, 27; March 1, 3.
T53	February 3, 12, 13, 14	February 15, 16, 17, 19, 21, 23, 24, 25, 27; March 1, 3.

In this experiment two doses of atabrine were given before infection and one dose five hours afterwards. The amount given was the maximum dose that would not cause toxic symptoms in the canary. The atabrine given in this way obviously did not prevent malaria. But it apparently had a measurable lethal or inhibitory effect on the sporozoites, for although the immediate death rate in the birds receiving atabrine was 60 per cent, in the controls not protected it was 80 per cent. The prepatent periods in the two surviving birds which received atabrine were six and eight days, respectively. In the three surviving control birds these periods were six, eight, and ten days, respectively. The atabrine apparently had no effect on this period in this experiment.

EXPERIMENT II

Birds T54, 55, and 56 were used in this experiment. February 9 each received 0.15 cc of the same sporozoite-saline mixture used in Experiment I. This was injected into the right pectoral muscle. A minute or two later birds T55 and T56 received 0.0005 gram of atabrine injected into the left pectoral muscle in 0.1 cc of distilled water. These two birds that received atabrine died February 10, the day after infection. T54 died the following day. These birds were overwhelmed by the infection, as were a majority of the birds in the first experiment. The single dose of atabrine had no favorable influence.

EXPERIMENT III

In order to prove that sudden death in birds receiving large doses of sporozoites was not due to any other constituent of the macerated thorax tissue of *Culex* mosquitoes, birds T67 and T68 were given 0.2 cc of a saline-thorax mixture into pectoral muscles February 10. This mixture was prepared by macerating the thorax tissue of twenty-five mosquitoes in 1 cc of saline solution. These mosquitoes were of the same strain as those used in the first two experiments. Each bird received the equivalent of the total thorax tissue of five mosquitoes. This was as large a dose as any used in the atabrine experiments. These birds were alive and negative March 22. They showed no ill effects whatever from the injection of uninfected thorax tissue of *Culex* mosquitoes.

EXPERIMENT IV

In this experiment ten birds were used, T57 to T66. Birds T57, 58, 59, 60, and 61 each received 0.0005 gram of atabrine intramuscularly February 10 and 11 and twice February 12. Thirty minutes after the last dose February 12 each of birds T57 to T66 received intramuscularly 0.2 cc of a saline-sporozoite mixture. The thorax tissue from 75 mosquitoes had been macerated with 3 cc saline so that 0.2 cc was equivalent to the thorax contents of 5 mosquitoes. Previous dissections from this lot of mosquitoes had shown a positive gland index of 20 per cent.

The following casualties occurred:

February 15: Birds T60, 62, 63, 64, and 65 died.

February 28: Bird T58 died.

March 1: Bird T66 died.

March 11: Bird T59 died.

Birds T57 and T61 were still alive March 22. Each of the five birds surviving beyond the normal prepatent period became infected as follows:

Bird No.	Blood-smear examinations.	
	Negative.	Positive.
T57	February 10, 17, 18, 19, 20	February 21, 23, 24, 25, 26, 27, 28; March 1, 2, 3, 4, 5.
T58	February 10, 17, 18, 19	February 20, 21, 23, 24, 25, 26, 27.
T59	February 10, 17, 18, 19, 20	February 21, 23, 24, 25, 26, 27, 28; March 1, 2, 3, 4, 5.
T61	February 10, 17, 18, 19, 20, 21	February 23, 24, 25, 26, 27, 28; March 1, 2, 3, 4, 5.
T66	February 10, 15, 17, 18, 19, 20, 21	February 23, 24, 25, 26, 27, 28.

In this experiment the immediate mortality among the birds receiving atabrine was 20 per cent, but among the control birds it was 80 per cent. The four doses of atabrine received prior to infection apparently had a measurably lethal or inhibitory effect on the sporozoites. But the atabrine did not prevent infection. The prepatent periods in the atabrine birds were 8, 9, 9, and 11 days.

In the one surviving control bird this prepatent period was eleven days. The course of the infection in the atabrine birds was of normal severity and duration, uninfluenced by the atabrine received prior to infection.

EXPERIMENT V

Ten birds were used in experiment V, T96–100, and 200–204. Birds T96, 97, 98, 99, and 100 each received 0.0005 gram of atabrine intramuscularly February 22, 23, 24, and 25. February 23 these birds and the controls 200–204 each received 0.1 cc of a saline-sporozoite mixture. This dose was equivalent to the thorax contents of 2.5 mosquitoes. Dissections had shown a positive salivary gland index of 40 per cent. The only immediate casualty was bird T96 which died February 25. Bird T100 died March 20. All the other birds were alive March 22.

All of the birds, excepting T96 which died, became infected as follows:

Bird No.	Blood-smear examinations.	
	Negative.	Positive.
T97	February 21, March 1	March 3, 5, 7, 9, 11, 13.
T98	February 21, March 2	March 4, 6, 8, 10, 12.
T99	February 21	March 1, 3, 5, 7, 9, 11, 13.
T100	February 21	March 2, 4, 6, 8, 10, 12.
200	February 21	March 2, 4, 6, 8, 10, 12.
201	February 21, March 1	March 3, 5, 7, 9, 11, 13.
202	February 21, March 2, 4, 6	March 8, 10, 12.
203	February 21	March 1, 2, 5, 7, 9, 11, 13.
204	February 21, March 2	March 4, 6, 8, 10, 12.

In this experiment the birds became infected, although smaller doses of sporozoites were injected and although the atabrine was continued for two days after the infection. The prepatent periods, and the severity and duration of the disease, appeared to have been uninfluenced by the atabrine.

EXPERIMENT VI

In experiment VI ten birds were used, numbers 205–214. Birds 205–209 each received 0.0005 gram atabrine intramuscularly February 22, 23, 24, and 25. During the night of February 23–24 they were all exposed to the bites of *Culex* mosquitoes as follows:

Cage.	Birds.	Mosquitoes.				
		Put in cage.	Recovered in morning.		Dissected.[a]	
			Total.	Blooded.	Total.	Positive salivary glands.
A	205,210	20	16	1	1	0
B	206,211	20	19	7	6	6
C	207,212	20	16	2	2	2
D	208,213	20	20	8	6	3
E	209,214	20	10	4	4	1

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[a] The dissection rates refer to the blooded mosquitoes. The birds devoured some of the mosquitoes—probably sluggish blooded ones.

The results of blood-smear examinations in these cases were as follows:

Bird No.	Blood-smear examinations.	
	Negative.	Positive.
205	February 21; March 1, 2, 5, 7, 9, 11	
206	February 21; March 2	March 4, 6, 8, 10, 12.
207	February 21; March 1	March 3, 5, 7, 9.
208	February 21; March 2, 4, 6	March 8, 10, 12.
209	February 21; March 1, 3, 5, 7, 9, 11	
210	February 21; March 2, 4, 6, 8, 10	
211	February 21; March 1	March 3, 5, 7, 9, 11, 13.
212	February 21; March 2, 4, 6, 8, 10	
213	February 21; March 1	March 3, 5, 7, 9, 11, 13.
214	February 21; March 2, 4	March 8, 10, 12, 14.

Bird 207 died March 10, bird 208 died March 13, and birds 206 and 214 died March 17.

Birds 205, 209, 210, and 212, which remained negative, were all proved to be susceptible birds by injection from an infected bird having the same strain of parasites.

In analyzing these results it is noted that birds 205, 209, 210, and 212 did not become infected. Of these birds, 205 and 210 were together in Cage A. Only one blooded mosquito was recovered from this cage, and it was negative on dissection. Therefore, it is not surprising that these two birds remained negative. Bird 209 had received atabrine. It was in Cage E and its control bird, No. 214, became positive. But of the four blooded mosquitoes recovered only one was found to be infected so it is possible that bird 209 was not bitten by an infected mosquito. The other negative bird was No. 212, which had not received atabrine. Two infected blooded mosquitoes were recovered from its Cage C. These may both have bitten bird 207, which shared the cage with 212 and became positive.

In other words the negative birds in this experiment have no significance as regards atabrine. But birds 206, 207, and 208 each received atabrine yet became infected. Bird 206 may have received a heavy infection as there were six infected blooded mosquitoes recovered from its cage. But birds 207 and 208 did not receive heavy infections. In these cases atabrine obviously did not prevent natural mosquito-borne infections.

EXPERIMENT VII

In this experiment ten birds were used, 223–232. March 30 and again March 21, at about 9 a. m. birds 223–227 each received an intramuscular injection of 0.00067 gram atabrine in 0.1 cc distilled water. March 31 at about 9.15 a. m. birds 223–232 each received 0.1 cc of a mixture of saline solution and macerated thorax tissue from infected *Culex* mosquitoes. A total of 75 mosquitoes and 4 cc of saline were used, so that each bird received the equivalent of the total thorax contents of 1.85 mosquitoes. The sporozoite index of the lot of mosquitoes was only 25 per cent. An examination of the mixture remaining after injection revealed a very few motile sporozoites. April 26 birds 227 and 228 died.

The following is the record of the blood-smear examinations of these birds:

Bird No.	Blood-smear examinations.	
	Negative.	Positive.
223	March 29; April 6, 9, 11	April 13, 15, 17.
224	March 29; April 7, 10	April 12, 14, 16, 18.
225	March 29; April 6, 9, 11, 13, 15, 17	
226	March 29; April 7, 10.	April 12, 14, 16, 18.
227	March 29; April 6, 9, 11, 13, 15, 17	
228	March 29; April 7	April 10, 12, 14, 16, 18.
229	March 29; April 6, 9, 11	April 13, 15, 17.
230	March 29; April 7	April 10, 12, 14, 16, 18.
231	March 29; April 6, 9, 11	April 13, 15, 17.
232	March 29; April 7, 10, 12, 14, 16, 18	

It will be seen that of the birds receiving atabrine, three (223, 224, 226) became infected and two (225, 227) did not, while of those not receiving atabrine four (228, 229, 230, 231) became infected and one (232) did not. The three negative birds were injected April 18 with blood containing the same strain of plasmodia. Bird 227 became positive April 22, bird 232 April 25, and bird 225 April 26. All had acute infections, from which it is assumed that they were not infected by the previous sporozoite injections.

In this experiment larger doses of atabrine and smaller doses of sporozoites were used. Rozeboom and Shah(16) have recently reported that they were unable to infect canaries with injections of less than 200 sporozoites. In other words, although theoretically one viable sporozoite should, perhaps, be able to infect a canary, yet more than 200 are required. In our experiment the sporozoites were not counted but by inspection it was known that the numbers were small. Therefore, it is not surprising that some of our birds did not become infected. Moreover, it cannot be called significant that although two of the atabrine birds remained negative only one of the control birds failed to be infected.

Here then atabrine in relatively large doses given before infection failed to protect birds receiving minimal doses of sporozoites.

SUMMARY

In a series of experiments to test the prophylactic action of atabrine in sporozoite infections of avian malaria it was found that although atabrine appears to have some lethal or inhibitory action on the sporozoites of avian malaria, it is but a true casual prophylactic.

CONCLUSIONS

1. Published reports indicate that atabrine given just before and for several days after mosquito-borne infection of human malaria will postpone, and in some cases, but not in others, will prevent entirely the appearance of malaria. Whether or not the atabrine has any effect on sporozoites is not clear from any published reports. Those experiments published suggest an early therapeutic effect with clinical rather than true prophylaxis.

2. In the experiments herewith reported, atabrine did not prevent the appearance of malaria in birds receiving maximum nontoxic doses of the drug and varying doses of sporozoites injected intramuscularly.

3. Atabrine did not prevent the appearance of malaria in birds exposed to the bites of infected *Culex* mosquitoes.

4. Atabrine appeared to reduce the immediate mortality rate in birds receiving large doses of injected sporozoites.

5. A mixture of macerated thorax tissue and normal saline from uninfected *Culex* mosquitoes, in doses equivalent to the thorax tissue of five mosquitoes, is not toxic to canaries when injected intramuscularly.

6. Mixtures of macerated thorax tissue and normal saline from malaria-infected *Culex* mosquitoes, in doses equivalent to the thorax tissue of 2 to 4.5 mosquitoes, injected intramuscularly, may prove rapidly fatal to canaries.

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WEIGHTS OF VISCERAL ORGANS OF FILIPINOS IN DIFFERENT DISEASES[1]

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The present paper is the fifth of a series of studies on the weights of visceral organs in Filipinos. In previous papers[2] we presented the weights of normal organs classified by age, body weight, and body length in persons dying of accidents. In this paper we shall report the variations of the weights of visceral organs for a number of selected diseases. The materials used in these studies were taken from the necropsy records of the Department of Pathology and Bacteriology and the Department of Legal Medicine of the University of the Philippines.

Table 1 presents the weights of visceral organs of Filipino children in the different diseases included in our study. Due to the great variability of the weights of organs in children at different ages, we decided to classify them into four age groups; namely, 1 year and less, 2 to 5 years, 6 to 10 years, and 11 to 15 years. Furthermore, due to insufficient available data, we included in the table only the following diseases: Typhoid fever, bacillary dysentery, suppurative and tuberculous meningitis, pulmonary tuberculosis, infantile beriberi, cholera infantum, endocarditis, broncho-pneumonia, lobar pneumonia, and acute and chronic nephritis. For purposes of comparison we have also included in Table 1 the normal weights of organs in accident cases as determined in our previous studies, cited in footnote 2.

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[1] Read before the Conjoint Annual Convention of the Philippine Islands Medical Association and the Philippine Public Health Association, December 13, 1933.

[2] De Jesus, P. I., and W. de Leon. Studies on the weights of visceral organs in Filipinos, Philip. Journ. Sci. 52 (1933) 97–98. De Jesus, P. I., W. de Leon, and P. Anzures. Normal weights of visceral organs in adult Filipinos, t. c. 99–109. De Leon, W., Arturo Garcia, and P. I. de Jesus. Normal weights of visceral organs in Filipino children, t. c. 111–118. De Jesus, P. I., W. de Leon, and J. M. Ramos. Normal weights of visceral organs in Filipinos in relation to length and body weight, t. c. 119–129.

The cases in Table 1 included under each of the diseases above mentioned were selected from those having only one main cause of death. Those cases having secondary causes of death or suffering from some other concomitant disease which might in any way confuse the weights of organs were not included. Thus, if the main cause of death was pulmonary tuberculosis, and the same person was also found to be suffering from endocarditis or from malaria, the case was excluded from our table. The same procedure was followed in preparing Table 2, which is presented below.

In the preparation of Table 1 we noted that in children the individual weights of organs under one age group showed a great deal of variation. This can perhaps be explained by the wide range of age groupings in our table and by the marked differences in body weights of children under one age group. We regret that due to an insufficient number of cases we were unable to classify the children into more age groups and into different body-weight groups. Consequently, no definite conclusions can be offered in the majority of diseases in Table 1.

In Tables 1 and 2 the mean weight was determined by taking the arithmetical mean of all the individual observations. The maximum and minimum weights were determined by first plotting the individual observations. In the graph the maximum and minimum weights were taken only from the group about the mean; a few observations that were found distantly separated from the majority of cases were rejected. However, in certain diseases all cases were included in the computation irrespective of the amount of deviation from the mean. This exception was made in the spleen of malaria and typhoid, where the maximum and minimum weights were obtained from the highest and lowest individual observations. The same exception was made in recording the maximum and minimum weights of the heart in endocarditis, arteriosclerosis, and chronic nephritis; and of the liver in cirrhosis.

Table 2 presents the weights of visceral organs of adult Filipinos in the following diseases: Typhoid fever, malaria, Asiatic cholera, amœbic and bacillary dysenteries, suppurative meningitis, tetanus, chronic pulmonary tuberculosis, tuberculous pneumonia, tuberculous meningitis, generalized miliary tuberculosis, beriberi, acute and chronic endocarditis, arteriosclerosis, broncho-pneumonia, lobar pneumonia, portal cirrhosis, hypertrophic cirrhosis, acute nephritis, chronic interstitial nephritis, chronic parenchymatous nephritis, and septicæmia.

As in Table 1 it can be readily seen that the weights of pathological organs in the adult vary with different diseases, the deviation being marked in some cases. Due to lack of time and help we were unable to determine by statistical methods whether these variations were significant or not.

A glance at Table 2 will show that acute diseases in general cause an absolute increase in the weight of the visceral organs with the exception of the heart. This increase is particularly notable in the spleen. The relative increases of weight, however, differ with different diseases as shown in Table 3.

Table 3 shows the percentage increase or decrease in the weights of organs from normal as affected by pathological conditions. This table was computed from the preceding table by comparing the weights of the pathological organs and the weights of the organs in accident cases.

It is interesting to note in Table 3 that the weight of the heart is markedly increased in chronic endocarditis, arteriosclerosis, beriberi, and chronic interstitial nephritis. It is only slightly increased in lobar pneumonia and chronic parenchymatous nephritis. The weight remains almost unaffected in tetanus, suppurative meningitis, acute nephritis, and septicæmia. It is markedly decreased in typhoid fever and amœbic dysentery, and only slightly decreased in chronic pulmonary tuberculosis, tuberculous meningitis, tuberculous pneumonia, malaria, bacillary dysentery, portal cirrhosis, and broncho-pneumonia. In cholera, generalized miliary tuberculosis, acute endocarditis, and hypertrophic cirrhosis, where the number of observations was less than ten in each group, no definite conclusions can be offered.

In Table 3 it will also be observed that the weight of the spleen is considerably increased in malaria, portal cirrhosis, septicæmia, beriberi, typhoid fever, lobar pneumonia, acute nephritis, broncho-pneumonia, and suppurative meningitis. It is only slightly increased in chronic pulmonary tuberculosis, chronic endocarditis, chronic interstitial nephritis, chronic parenchymatous nephritis, bacillary dysentery, and amœbic dysentery. It is slightly decreased in tuberculous meningitis, arteriosclerosis, and tetanus. In the rest of the diseases included in our study the cases were too few to warrant definite conclusions.

TABLE 1.—*Cases and weights of visceral organs of Filipino children in certain diseases.*

Cause of death.	Sex.	Age.	Heart.				Spleen.				Liver.			
			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
				Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
		Yrs.		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Trauma	M	1 or <1	13	32.92	19	43	12	31.83	6	63	12	247.83	128	690
Do	M	2- 5	45	69.58	35	105	42	47.19	22	84	36	463.14	199	834
Do	M	6-10	69	105.64	65	155	67	58.09	21	127	56	642.59	398	1,144
Do	M	11-15	76	159.32	85	330	73	86.52	40	180	60	893.55	650	1,471
Do	F	1 or <1	8	33.75	19	59	9	22.67	10	39	8	202.00	97	335
Do	F	2- 5	35	64.94	42	106	37	42.51	23	80	30	418.50	267	716
Do	F	6-10	20	94.75	60	128	19	53.00	17	106	17	673.35	400	906
Do	F	11-15	17	167.00	117	232	17	104.53	57	215	15	865.60	650	1,429
Typhoid fever	M	1 or <1	1	61.00	------	------	1	31.00	------	------	1	257.00	------	------
Do	M	2- 5	1	60.00	------	------	1	77.00	------	------	1	461.00	------	------
Do	M	6-10	4	106.25	87	134	3	81.00	33	125	4	757.00	585	892
Do	M	11-15	7	152.29	87	242	7	165.00	95	317	7	1,053.57	645	1,437
Do	F	1 or <1	1	50.00	------	------	1	29.00	------	------	1	277.00	------	------
Do	F	2- 5	4	64.50	54	87	4	53.25	27	124	4	464.75	270	797
Do	F	6-10	7	82.86	73	94	8	82.00	37	145	7	662.00	505	752
Do	F	11-15	13	139.38	87	217	12	197.25	66	750	13	1,059.46	740	1,300
Bacillary dysentery	M	1 or <1	27	28.22	12	47	27	18.04	8	37	24	223.50	102	625
Do	M	2- 5	18	49.83	30	75	18	25.89	10	40	17	341.88	127	532
Do	M	6-10	3	80.33	62	104	3	40.33	20	78	3	555.00	500	612
Do	M	11-15	2	238.00	166	310	2	208.50	80	337	2	1,482.00	1,377	1,587
Do	F	1 or <1	14	29.93	12	50	14	15.79	8	29	12	213.50	95	355
Do	F	2- 5	16	51.94	35	85	15	26.80	15	43	15	365.87	185	595
Do	F	6-10	2	64.00	51	77	2	29.00	21	37	2	465.00	395	535

Do	F	11-15	4	120.50	97	135	4	62.75	21	92	4	941.00	697	1,210
Suppurative and tuberculous meningitis	M	1 or <1	17	38.12	16	64	17	22.71	7	48	16	232.50	47	365
Do	M	2- 5	7	54.71	35	72	7	36.14	24	56	7	334.00	221	471
Do	M	6-10	10	95.10	70	130	10	49.60	26	80	10	583.30	249	706
Do	M	11-15	10	154.60	106	241	10	78.00	32	115	11	839.18	447	1,436
Do	F	1 or <1	12	32.33	16	52	12	22.33	9	44	11	222.18	110	335
Do	F	2- 5	7	56.00	42	72	7	29.71	16	39	6	414.50	287	637
Do	F	6-10	8	88.38	36	127	8	52.50	26	130	7	545.14	330	800
Do	F	11-15	3	155.33	110	236	3	88.33	47	140	3	888.33	740	1,040
Pulmonary tuberculosis	M	1 or <1	14	26.29	17	39	12	23.83	12	50	15	249.40	151	455
Do	M	2- 5	41	45.29	27	82	40	34.60	15	75	38	331.21	204	532
Do	M	6-10	4	102.75	51	180	3	49.67	46	55	4	557.00	485	613
Do	M	11-15	10	139.30	90	185	10	86.10	55	167	10	798.40	562	1,350
Do	F	1 or <1	14	30.29	12	51	15	20.73	5	42	13	198.38	92	328
Do	F	2- 5	33	38.27	22	73	34	28.79	12	124	33	308.15	132	825
Do	F	6-10	5	92.00	59	120	6	44.83	24	54	6	612.50	327	864
Do	F	11-15	5	168.40	89	279	3	74.33	55	107	5	936.00	724	1,165
Infantile beriberi	M	1 or <1	26	36.50	17	60	28	16.64	9	38	28	161.93	105	254
Do	M	2- 5	2	86.50	46	127	1	29.00	-------	-------	1	480.00	-------	-------
Do	M	6-10	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------
Do	M	11-15	4	286.50	192	388	4	186.00	171	06	4	1,145.50	888	1,440
Do	F	1 or <1	19	34.26	23	58	17	15.29	6	57	18	160.17	28	231
Do	F	2- 5	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------
Do	F	6-10	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------
Do	F	11-15	1	252.00	-------	-------	1	209.00	-------	-------	1	1,375.00	-------	-------
Cholera infantum	M	1 or <1	27	22.59	8	40	27	13.70	5	27	27	140.56	41	249
Do	M	2- 5	1	25.00	-------	-------	1	24.00	-------	-------	1	252.00	-------	-------
Do	M	6-10	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------
Do	M	11-15	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------
Do	F	1 or <1	17	18.59	7	35	16	11.19	5	27	17	127.47	42	269
Do	F	2- 5	1	32.00	-------	-------	2	19.00	15	23	2	226.00	186	266
Do	F	6-10	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------
Do	F	11-15	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------	-------

TABLE 1.—*Cases and weights of visceral organs of Filipino children in certain diseases*—Continued.

Cause of death.	Sex.	Age.	Heart.				Spleen.				Liver.			
			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
				Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
	g.	*Yrs.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Endocarditis	M	1 or <1	1	52.00	------	------	1	15.00	------	------	1	182.00	------	------
Do	M	2- 5	1	66.00	------	------	1	23.00	------	------	1	370.00	------	------
Do	M	6-10	------	------	------	------	------	------	------	------	------	------	------	------
Do	M	11-15	3	472.33	300	644	3	82.00	72	94	2	1,249.50	1,150	1,349
Do	F	1 or <1	1	60.00	------	------	------	------	------	------	1	170.00	------	------
Do	F	2- 5	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	6-10	2	264.50	231	298	2	99.50	76	123	2	943.00	811	1,075
Do	F	11-15	7	335.43	232	583	7	94.29	35	125	7	1,256.43	723	2,732
Broncho-pneumonia	M	1 or <1	58	27.78	10	60	57	16.91	3	56	57	182.88	52	422
Do	M	2- 5	20	51.50	30	100	19	34.26	12	101	17	370.00	201	623
Do	M	6-10	2	208.50	78	339	8	65.00	34	93	3	546.00	298	790
Do	M	11-15	1	185.00	------	------	1	62.00	------	------	1	1,197.00	------	------
Do	F	1 or <1	44	26.43	7	57	45	14.96	3	52	45	178.98	28	375
Do	F	2- 5	11	50.18	21	82	9	41.22	10	75	9	340.44	157	560
Do	F	6-10	5	96.40	60	178	5	123.60	31	382	4	735.75	450	1,320
Do	F	11-15	1	200.00	------	------	1	112.00	------	------	1	688.00	------	------
Lobar pneumonia	M	1 or <1	6	29.67	12	42	6	25.33	4	58	6	229.00	68	374
Do	M	2- 5	4	47.75	28	65	4	31.00	18	46	4	330.25	186	485
Do	M	6-10	5	97.00	84	110	5	62.40	30	92	5	671.40	497	840
Do	M	11-15	3	147.67	131	170	3	483.67	68	970	3	1,159.67	1,015	1,271
Do	F	1 or <1	11	26.55	12	41	10	18.30	8	35	9	217.78	110	340
Do	F	2- 5	3	45.33	38	54	3	23.30	19	31	3	487.67	325	705
Do	F	6-10	1	82.00	------	------	1	49.00	------	------	1	600.00	------	------
Do	F	11-15	1	135.00	------	------	1	81.00	------	------	1	1,053.00	------	------

Acute nephritis	M	1 or <1	12	31.92	19	64	11	18.82	13	35	11	207.91	160	264
Do.	M	2–5												
Do.	M	6–10	2	113.50	87	140	2	69.00	58	80	2	648.50	462	835
Do.	M	11–15	1	204.00			1	110.00			1	1,210.00		
Do.	F	1 or <1	7	27.57	15	37	7	16.71	11	23	5	156.40	105	204
Do.	F	2–5	1	40.00			1	22.00			1	275.00		
Do.	F	6–10												
Do.	F	11–15	1	173.00			1	76.00			1	884.00		
Chronic nephritis	M	1 or <1												
Do.	M	2–5												
Do.	M	6–10	1	62.00			1	115.00			1	337.00		
Do.	M	11–15	6	237.50	92	320	6	101.67	65	140	5	917.20	760	1,150
Do.	F	1 or <1	1	22.00			1	22.00			1	100.00		
Do.	F	2–5	1	70.00			1	29.00			1	297.00		
Do.	F	6–10	1	94.00			1	215.00			1	580.00		
Do.	F	11–15												

TABLE 1.—*Cases and weights of visceral organs of Filipino children in certain diseases*—Continued.

Cause of death.	Sex.	Age.	Pancreas.				Suprarenals.							
							Both.				Right.			
			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
				Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
		Yrs.		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Trauma	M	1 or <1	2	11.00	7	15	8	6.50	4	11	5	3.60	3	4
Do	M	2-5	6	26.83	17	33	18	7.06	4	12	15	3.60	2	5
Do	M	6-10	18	47.50	20	76	35	9.57	4	15	24	4.88	2	7
Do	M	11-15	15	60.27	42	100	36	10.08	5	15	22	5.36	3	8
Do	F	1 or <1	2	17.00	15	19	6	6.33	3	10	2	4.00	3	5
Do	F	2-5	7	23.71	19	29	17	6.06	3	11	9	3.00	2	5
Do	F	6-10	7	47.00	30	57	11	8.64	4	14	9	4.44	3	7
Do	F	11-15	5	63.80	52	90	10	12.10	5	14	9	5.78	2	12
Typhoid fever	M	1 or <1	1	14.00	------	------	1	3.00	------	------	1	1.00	------	------
Do	M	2-5	------	------	------	------	1	12.00	------	------	1	6.00	------	------
Do	M	6-10	1	54.00	------	------	1	10.00	------	------	------	------	------	------
Do	M	11-15	------	------	------	------	4	10.50	8	12	------	------	------	------
Do	F	1 or <1	------	------	------	------	1	5.00	------	------	------	------	------	------
Do	F	2-5	1	17.00	------	------	4	8.25	5	10	1	5.00	------	------
Do	F	6-10	2	41.00	37	45	3	10.33	9	13	2	4.50	4	5
Do	F	11-15	1	87.00	------	------	9	10.22	7	14	1	6.00	------	------
Bacillary dysentery	M	1 or <1	7	7.71	5	10	16	5.06	2	9	8	2.63	2	5
Do	M	2-5	3	22.00	14	27	13	6.46	4	10	3	4.00	2	5
Do	M	6-10	------	------	------	------	3	8.00	7	9	------	------	------	------
Do	M	11-15	------	------	------	------	1	13.00	------	------	------	------	------	------
Do	F	1 or <1	3	12.00	7	18	9	5.55	2	12	2	4.00	2	6
Do	F	2-5	6	20.67	7	32	12	7.08	2	13	5	4.80	4	7

Do	F	6–10					1	10.00			1	5.00		
Do	F	11–15					3	12.33	11	15				
Suppurative and tuberculous meningitis	M	1 or <1	7	8.86	4	11	11	6.00	8	8	6	4.00	8	5
Do	M	2– 5	3	17.33	13	26	5	6.20	4	9	3	3.33	2	5
Do	M	6–10	4	35.25	17	52	8	9.25	5	14	3	3.67	3	4
Do	M	11–15	2	42.00	35	49	6	11.50	9	15	1	4.00		
Do	F	1 or <1	1	17.00			7	6.86	4	12	1	4.00		
Do	F	2– 5	4	23.75	14	32	5	7.00	5	12	4	3.50	2	6
Do	F	6–10	3	15.00	7	36	3	8.83	7	10	1	5.00		
Do	F	11–15					1	17.00			1	9.00		
Pulmonary tuberculosis	M	1 or <1	2	12.50	6	19	7	4.71	2	8	3	4.33	3	6
Do	M	2– 5	9	17.11	9	24	26	6.77	3	12	13	3.31	2	6
Do	M	6–10	1	62.00			2	5.00	4	6				
Do	M	11–15	1	47.00			5	12.60	10	18	1	9.00		
Do	F	1 or <1					6	4.83	2	7				
Do	F	2– 5	8	15.63	7	28	22	5.00	2	12	8	3.25	2	6
Do	F	6–10	2	33.50	4	43	4	6.00	4	8	1	3.00		
Do	F	11–15	2	51.00	2	60	2	12.00	9	15				
Infantile beriberi	M	1 or <1	7	6.86	3	11	17	4.88	2	10	8	2.63	1	5
Do	M	2– 5												
Do	M	6–10												
Do	M	11–15												
Do	F	1 or <1	3	8.67	7	10	9	6.33	3	11	3	4.67	3	7
Do	F	2– 5												
Do	F	6–10												
Do	F	11–15												
Cholera infantum	M	1 or <1	1	12.00			14	4.71	3	7	3	2.67	2	4
Do	M	2– 5												
Do	M	6–10												
Do	M	11–15												
Do	F	1 or <1	2	8.50	8	9	13	4.62	2	10	4	3.25	2	5
Do	F	2– 5												
Do	F	6–10												
Do	F	11–15												

TABLE 1.—*Cases and weights of visceral organs of Filipino children in certain diseases*—Continued.

Cause of death.	Sex.	Age.	Pancreas.				Suprarenals.							
							Both.				Right.			
			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
				Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
		Yrs.		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Endocarditis	M	1 or <1												
Do	M	2- 5	1	23.00			1	16.00			1	7.00		
Do	M	6-10												
Do	M	11-15	2	106.50	78	135	3	13.00	11	15	2	5.50	5	6
Do	F	1 or <1												
Do	F	2- 5												
Do	F	6-10												
Do	F	11-15	1	86.00			5	10.80	7	14	1	7.00		
Broncho-pneumonia	M	1 or <1	12	9.58	2	20	34	5.71	2	11	13	3.31	1	6
Do	M	2- 5	8	14.25	8	27	11	5.27	4	6	8	2.38	2	3
Do	M	6-10												
Do	M	11-15												
Do	F	1 or <1	13	7.54	3	14	24	4.63	2	9	10	2.40	1	4
Do	F	2- 5	2	17.50	15	20	6	5.83	3	9	2	3.00	2	4
Do	F	6-10	2	40.00	34	46	4	7.00	4	11	2	3.50	2	5
Do	F	11-15												
Lobar pneumonia	M	1 or <1					3	5.00	3	6				
Do	M	2- 5					3	4.67	3	6				
Do	M	6-10					3	8.33	7	9				
Do	M	11-15	1	70.00			1	17.00						
Do	F	1 or <1	1	8.00			4	5.75	5	7				

Do	F	2- 5					1	5.00						
Do	F	6-10	1	37.00			1	14.00						
Do	F	11-15	1	68.00			1	10.00			1	5.00		
Acute nephritis	M	1 or <1					3	4.33	8	5	1	2.00		
Do	M	2- 5												
Do	M	6-10	1	62.00			1	10.00			1	5.00		
Do	M	11-15					1	12.00						
Do	F	1 or <1	1	9.00			2	4.50	3	6	2	2.00	1	9
Do	F	2- 5					1	3.00						
Do	F	6-10												
Do	F	11-15	1	55.00			1	11.00			1	5.00		
Chronic nephritis	M	1 or <1												
Do	M	2- 5												
Do	M	6-10					1	7.00						
Do	M	11-15	1	112.00			4	12.75	8	22	1	10.00		
Do	F	1 or <1					1	7.00						
Do	F	2- 5												
Do	F	6-10					1	10.00						
Do	F	11-15												

TABLE 1.—*Cases and weights of visceral organs of Filipino children in certain diseases*—Continued.

Cause of death.	Sex.	Age.	Suprarenals.				Kidneys.											
			Left.				Both.				Right.				Left.			
			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
				Mean.	Mini-mum.	Maxi-mum.		Mean.	Mini-mum.	Maxi-mum.		Mean.	Mini-mum.	Maxi-mum.		Mean.	Mini-mum.	Maxi-mum.
		Yrs.		g.	g.	g.		g.	g.	g.		g.	g.	g.		g.	g.	g.
Trauma	M	1 or <1	5	4.00	3	7	11	53.36	26	105	4	32.50	18	50	4	35.25	22	55
Do	M	2- 5	15	3.60	2	5	36	82.78	53	137	29	39.28	23	60	29	41.59	30	77
Do	M	6-10	24	5.21	2	9	59	121.93	75	286	40	59.48	40	143	40	60.88	35	143
Do	M	11-15	22	6.18	3	14	66	167.58	100	294	47	81.77	39	147	47	85.66	50	147
Do	F	1 or <1	2	4.00	3	5	8	45.25	20	67	2	28.50	25	32	2	32.50	30	35
Do	F	2- 5	9	3.56	2	6	27	81.41	54	122	19	38.32	25	60	19	39.16	27	62
Do	F	6-10	9	4.78	3	7	18	110.61	74	174	14	53.57	35	87	14	57.00	38	90
Do	F	11-15	9	6.78	3	11	15	173.73	110	232	13	83.23	55	112	13	88.15	55	120
Typhoid fever	M	1 or <1	1	2.00	------	------	1	71.00	------	------	1	36.00	------	------	1	35.00	------	------
Do	M	2- 5	1	6.00	------	------	1	133.00	------	------	1	65.00	------	------	1	68.00	------	------
Do	M	6-10	------	------	------	------	4	159.00	136	189	------	------	------	------	------	------	------	------
Do	M	11-15	------	------	------	------	7	201.29	149	320	------	------	------	------	------	------	------	------
Do	F	1 or <1	------	------	------	------	1	72.00	------	------	------	------	------	------	------	------	------	------
Do	F	2- 5	1	4.00	------	------	4	83.50	60	132	1	30.00	------	------	1	30.00	------	------
Do	F	6-10	2	6.50	5	8	7	141.86	108	266	3	83.33	58	133	3	86.33	62	133
Do	F	11-15	1	7.00	------	------	12	191.08	142	237	2	121.00	105	137	2	109.50	100	119
Bacillary dysentery	M	1 or <1	8	2.75	2	4	25	46.96	23	102	7	25.43	11	51	7	25.71	12	51
Do	M	2- 5	8	4.00	2	5	16	70.75	35	122	1	39.00	------	------	1	40.00	------	------

Do	M	6–10	------	------	------	------	3	111.00	103	117	------	------	------	------	------	------	------	------
Do	M	11–15	------	------	------	------	2	207.00	188	226	------	------	------	------	------	------	------	------
Do	F	1 or <1	2	4.50	3	6	15	47.80	21	82	4	22.00	11	31	4	22.75	11	31
Do	F	2– 5	5	5.40	4	9	16	94.81	55	186	7	56.57	30	91	7	59.29	30	95
Do	F	6–10	1	5.00	------	------	2	102.50	69	136	------	------	------	------	------	------	------	------
Do	F	11–15	------	------	------	------	4	167.75	131	210	------	------	------	------	------	------	------	------
Suppurative and tuberculous meningitis	M	or <1	6	4.00	3	5	16	54.81	24	85	6	18.67	12	27	6	21.17	12	31
Do	M	2– 5	3	3.67	3	4	6	73.17	50	92	3	31.00	27	37	3	29.00	23	37
Do	M	6–10	3	5.33	3	7	10	123.00	87	179	3	61.00	54	66	3	67.67	59	74
Do	M	11–15	1	5.00	------	------	10	180.50	113	275	1	59.00	------	------	1	54.00	------	------
Do	F	1 or <1	1	6.00	------	------	11	49.64	20	83	2	30.50	27	34	2	33.00	28	38
Do	F	2– 5	4	4.00	3	6	7	95.14	71	139	4	49.00	39	59	4	54.50	40	82
Do	F	6–10	1	5.00	------	------	6	109.67	100	122	1	51.00	------	------	1	59.00	------	------
Do	F	11–15	1	8.00	------	------	4	194.75	145	236	2	96.00	89	103	2	111.00	89	133
Pulmonary tuberculosis	M	1 or <1	3	4.33	3	6	12	46.08	24	60	3	26.33	26	27	3	28.00	26	29
Do	M	2– 5	13	3.62	2	6	34	70.79	40	117	11	31.27	25	44	11	32.73	23	45
Do	M	6–10	------	------	------	------	3	126.67	99	170	------	------	------	------	------	------	------	------
Do	M	11–15	1	9.00	------	------	9	189.33	117	236	1	103.00	------	------	1	133.00	------	------
Do	F	1 or <1	------	------	------	------	10	44.00	24	66	------	------	------	------	------	------	------	------
Do	F	2– 5	8	3.25	2	6	32	63.22	33	118	8	28.88	17	35	8	27.88	19	33
Do	F	6–10	1	3.00	------	------	7	110.00	40	140	1	59.00	------	------	1	63.00	------	------
Do	F	11–15	------	------	------	------	7	196.71	62	311	1	87.00	------	------	1	76.00	------	------
Infantile beriberi	M	1 or <1	8	2.75	1	5	28	38.54	25	55	12	17.75	12	26	12	18.83	12	26
Do	M	2– 5	------	------	------	------	1	112.00	------	------	------	------	------	------	------	------	------	------
Do	M	6–10	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	M	11–15	------	------	------	------	2	217.50	162	273	------	------	------	------	------	------	------	------
Do	F	1 or <1	3	4.33	4	5	17	39.18	25	64	6	20.50	17	26	6	21.00	16	25
Do	F	2– 5	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	6–10	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	11–15	------	------	------	------	1	202.00	------	------	------	------	------	------	------	------	------	------

TABLE 1.—*Cases and weights of visceral organs of Filipino children in certain diseases*—Continued.

Cause of death.	Sex.	Age.	Suprarenals.				Kidneys.											
			Left.				Both.				Right.				Left.			
			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
				Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
		Yrs.		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Cholera infantum	M	1 or <1	3	2.33	2	3	25	35.80	15	61	2	26.00	24	28	2	25.50	22	29
Do	M	2- 5	------	------	------	------	1	55.00	------	------	------	------	------	------	------	------	------	------
Do	M	6-10	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	M	11-15	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	1 or <1	4	3.25	2	5	17	35.06	14	56	5	17.40	13	21	5	17.60	15	20
Do	F	2- 5	------	------	------	------	2	59.00	43	75	------	------	------	------	------	------	------	------
Do	F	6-10	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	11-15	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Endocarditis	M	1 or <1	------	------	------	------	1	45.00	------	------	------	------	------	------	------	------	------	------
Do	M	2- 5	1	9.00	------	------	1	80.00	------	------	1	40.00	------	------	1	40.00	------	------
Do	M	6-10	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	M	11-15	2	6.50	6	7	3	213.33	210	216	2	103.50	101	106	2	109.50	109	110
Do	F	1 or <1	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	2- 5	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	6-10	------	------	------	------	2	199.00	146	252	------	------	------	------	------	------	------	------
Do	F	11-15	1	7.00	------	------	7	207.00	164	259	2	117.50	109	126	2	109.00	103	115
Broncho-pneumonia	M	1 or <1	13	3.38	1	5	56	39.00	13	74	17	23.00	8	37	17	21.82	7	34
Do	M	2- 5	8	2.88	2	4	18	79.78	41	164	10	38.40	25	60	10	37.00	24	57
Do	M	6-10	------	------	------	------	3	110.00	60	150	1	30.00	------	------	1	30.00	------	------
Do	M	11-15	------	------	------	------	1	205.00	------	------	------	------	------	------	------	------	------	------
Do	F	1 or <1	10	2.80	2	5	44	37.23	10	92	9	20.67	8	30	9	21.33	8	33

Do	F	2–5	2	3.00	2	4	9	68.67	21	93	5	38.20	28	47	5	39.00	27	48
Do	F	6–10	2	4.00	2	6	5	136.00	88	275	2	46.00	37	55	2	57.50	52	63
Do	F	11–15	------	------	------	------	1	94.00	------	------	------	------	------	------	------	------	------	------
Lobar pneumonia	M	1 or <1	------	------	------	------	6	56.50	38	86	1	22.00	------	------	1	22.00	------	------
Do	M	2–5	------	------	------	------	5	74.40	47	95	1	25.00	------	------	1	22.00	------	------
Do	M	6–10	------	------	------	------	5	132.20	120	157	------	------	------	------	------	------	------	------
Do	M	11–15	------	------	------	------	3	201.00	154	232	------	------	------	------	------	------	------	------
Do	F	1 or <1	------	------	------	------	9	47.22	22	68	------	------	------	------	------	------	------	------
Do	F	2–5	------	------	------	------	2	67.50	55	80	------	------	------	------	------	------	------	------
Do	F	6–10	------	------	------	------	1	137.00	------	------	------	------	------	------	------	------	------	------
Do	F	11–15	1	5.00	------	------	1	208.00	------	------	1	98.00	------	------	1	110.00	------	------
Acute nephritis	M	1 or <1	1	3.00	------	------	11	52.27	32	115	1	22.00	------	------	1	20.00	------	------
Do	M	2–5	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	M	6–10	1	5.00	------	------	2	145.50	97	194	1	94.00	------	------	1	100.00	------	------
Do	M	11–15	------	------	------	------	1	224.00	------	------	------	------	------	------	------	------	------	------
Do	F	1 or <1	2	2.50	2	3	7	42.14	25	56	5	19.00	12	27	5	19.60	13	29
Do	F	2–5	------	------	------	------	1	65.00	------	------	------	------	------	------	------	------	------	------
Do	F	6–10	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	F	11–15	1	6.00	------	------	1	281.00	------	------	1	144.00	------	------	1	137.00	------	------
Chronic nephritis	M	1 or <1	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	M	2–5	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Do	M	6–10	------	------	------	------	1	110.00	------	------	------	------	------	------	------	------	------	------
Do	M	11–15	1	12.00	------	------	5	203.40	170	282	1	135.00	------	------	1	147.00	------	------
Do	F	1 or <1	------	------	------	------	1	45.00	------	------	------	------	------	------	------	------	------	------
Do	F	2–5	------	------	------	------	1	90.00	------	------	------	------	------	------	------	------	------	------
Do	F	6–10	------	------	------	------	1	184.00	------	------	------	------	------	------	------	------	------	------
Do	F	11–15	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------

TABLE 2.—*Cases and weights of visceral organs of adult Filipinos in certain diseases.*

Cause of death and sex.	Heart.				Spleen.				Liver.			
	Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
Trauma:		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Male	620	278.11	230	330	614	116.82	50	180	494	1,211.04	900	1,500
Female	148	237.18	170	310	147	91.84	40	140	125	1,090.64	700	1,500
Typhoid fever:												
Male	140	215.23	160	300	129	217.31	68	983	137	1,358.13	760	1,840
Female	57	193.00	150	225	56	192.73	81	515	49	1,245.96	890	1,910
Malaria:												
Male	47	263.23	200	300	45	402.00	130	1,870	46	1,262.17	1,040	2,150
Female	9	221.78	160	280	9	270.56	130	330	7	1,486.29	860	2,160
Asiatic cholera:												
Male	8	273.75	220	350	8	142.25	50	210	8	1,327.25	1,020	1,580
Female	4	266.50	205	345	5	68.20	30	130	4	1,186.25	890	1,470
Amœbic dysentery:												
Male	45	219.36	165	260	43	118.14	40	220	39	1,338.74	730	2,520
Female	18	196.17	150	260	17	95.76	45	160	17	1,344.18	880	1,830
Bacillary dysentery:												
Male	19	236.53	190	280	17	113.29	50	170	18	1,309.67	880	1,650
Female	15	229.73	160	290	15	132.33	40	170	15	1,400.20	830	2,020
Suppurative meningitis:												
Male	34	262.47	200	315	27	159.56	70	270	33	1,380.45	830	2,160
Female	19	267.16	200	350	16	123.81	40	250	19	1,272.84	870	1,650
Tetanus:												
Male	16	265.81	210	300	14	103.14	45	150	13	1,142.54	940	1,500
Female	6	257.00	200	300	6	86.17	40	150	5	1,172.60	930	1,620

Chronic pulmonary tuberculosis:											
Male	218	230.80	155	330	213	132.31	50	270	220	1,154.50	620	2,680
Female	102	203.26	140	300	92	106.24	40	210	85	1,074.60	560	1,830
Tuberculous pneumonia:												
Male	11	209.18	170	270	10	129.80	50	200	8	1,168.38	850	1,600
Female	4	205.00	180	240	3	96.33	85	120	4	1,096.75	770	1,280
Tuberculous meningitis:												
Male	13	227.08	180	280	13	89.00	30	160	13	1,015.92	830	1,440
Female	8	190.13	160	230	8	67.25	40	95	8	905.25	690	1,020
General miliary tuberculosis:												
Male	8	226.25	160	290	8	169.38	70	260	8	1,336.75	1,010	1,550
Female	3	165.00	120	230	3	135.00	100	205	3	1,137.33	840	1,530
Beriberi:												
Male	38	348.68	280	410	36	249.64	120	420	33	1,330 67	700	1,750
Female	10	316.60	240	380	10	188.00	90	300	7	1,387.86	1,160	1,770
Acute endocarditis:												
Male	3	302.83	280	320	1	288.00	----------	----------	2	1,444.50	1,389	1,500
Female	6	297.67	220	430	5	146.00	70	290	5	1,391.80	990	1,830
Chronic endocarditis:												
Male	52	405.67	270	890	46	156.59	60	290	49	1,338.88	850	1,980
Female	43	347.95	200	740	40	109.30	30	210	41	1,199.88	780	1,890
Arteriosclerosis:												
Male	41	386.12	250	750	38	115.74	50	250	35	1.329.17	800	2,140
Female	29	330.03	200	600	29	88.34	30	160	27	1,158.59	630	1,620
Broncho-pneumonia:												
Male	37	245.43	200	300	34	163.41	65	280	36	1,304.94	730	2,010
Female	25	234.80	190	290	21	121.29	60	190	23	1,170.13	580	1,550
Lobar pneumonia:												
Male	99	285.76	235	335	97	217.70	80	300	95	1,481.46	860	2,230
Female	40	261.75	190	300	36	180.94	55	260	39	1,312.64	660	2,020
Portal cirrhosis:												
Male	29	250.93	170	330	28	238.30	90	380	26	1,034.12	560	2,100
Female	7	240.71	180	310	6	202.83	85	300	7	1,078.43	660	1,720

TABLE 2.—*Cases and weights of visceral organs of adult Filipinos in certain diseases*—Continued.

Cause of death and sex.	Heart.				Spleen.				Liver.			
	Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
		g.	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Hypertrophic cirrhosis:												
Male	4	218.50	150	280	3	618.00	425	845	5	1,563.40	1,110	2,990
Female	3	253.67	170	330	3	282.33	270	295	3	1,143.00	930	1,500
Acute nephritis:												
Male	8	301.25	240	360	7	165.43	60	290	7	1,214.43	930	1,400
Female	18	234.67	165	310	16	145.38	70	250	18	1,326.11	850	2,240
Chronic interstitial nephritis:												
Male	56	336.43	200	650	53	132.75	55	230	51	1,254.25	620	2,190
Female	33	313.64	170	650	30	96.90	25	160	33	1,109.88	520	1,690
Chronic parenchymatous nephritis:												
Male	13	287.31	200	380	11	138.36	50	240	12	1,200.67	860	1,480
Female	19	279.68	215	370	16	128.13	50	250	18	1,323.22	500	1,810
Septicæmia:												
Male	33	270.00	210	340	33	206.79	100	350	31	1,492.42	1,030	2,150
Female	27	240.15	190	290	27	235.19	100	400	26	1,477.35	950	1,990

TABLE 2.—*Cases and weights of visceral organs of adult Filipinos in certain diseases*—Continued.

Cause of death and sex.	Pancreas.				Suprarenals.							
					Both.				Right.			
	Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
		g.	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Trauma:												
Male	121	105.88	55	155	287	15.96	12	20	187	8.35	6	11
Female	39	86.23	50	120	84	14.57	10	19	52	7.06	4	10
Typhoid fever:												
Male	27	95.37	60	130	82	15.23	7	29	27	8.11	4	17
Female	15	93.53	55	130	33	14.64	9	21	12	7.58	4	12
Malaria:												
Male	14	109.93	80	160	20	17.00	10	29	15	8.20	5	12
Female	1	97.00	-------	-------	2	12.50	12	13	1	6.00	-------	-------
Asiatic cholera:												
Male	-------	-------	-------	-------	1	10.00	-------	-------	-------	-------	-------	-------
Female	-------	-------	-------	-------	1	10.00	-------	-------	-------	-------	-------	-------
Amœbic dysentery:												
Male	6	106.50	80	150	23	13.48	10	24	5	8.00	5	12
Female	3	77.67	60	95	9	14.56	8	22	4	5.20	4	7
Bacillary dysentery:												
Male	5	79.40	70	90	10	15.30	10	27	2	5.50	5	6
Female	2	82.00	69	95	8	15.63	10	22	3	7.67	5	10
Suppurative meningitis:												
Male	8	102.38	80	140	21	17.57	10	30	8	10.75	7	17
Female	4	85.00	70	110	10	16.90	9	25	4	10.00	7	17

TABLE 2.—*Cases and weights of visceral organs of adult Filipinos in certain diseases*—Continued.

Cause of death and sex.	Pancreas.				Suprarenals.							
					Both.				Right.			
	Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
		Mean.	Mini-mum.	Mini-mum.		Mean.	Mini-mum.	Maxi-mum.		Mean.	Mini-mum.	Maxi mum.
Tetanus:		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Male					5	17.40	16	19	5	8.20	7	9
Female	1	107.00			1	24.00			1	10.00		
Chronic pulmonary tuberculosis:												
Male	41	98.22	65	130	135	14.19	7	28	41	7.34	4	12
Female	13	79.92	60	120	55	12.76	6	26	14	7.14	4	14
Tuberculous pneumonia:												
Male	2	100.00	70	130	3	14.00	11	19	3	7.00	5	9
Female					3	14.00	12	16				
Tuberculous meningitis:												
Male	1	71.00			5	13.40	7	22	1	8.00		
Female	2	107.50	95	120	7	14.71	12	20	1	8.00		
General miliary tuberculosis:												
Male					7	14.57	11	20	1	6.00		
Female	1	70.00			2	17.00	15	19	1	7.00		
Beriberi:												
Male	11	114.82	90	140	19	14.89	10	29	11	7.18	5	12
Female	3	119.33	90	150	7	16.29	10	20	4	10.50	10	11
Acute endocarditis:												
Male	1	165.00			2	12.50	11	14	1	6.00		
Female					3	14.00	11	19				

Chronic endocarditis:												
Male	10	114.80	80	155	32	16.06	10	25	10	8.60	6	16
Female	8	108.00	75	160	19	12.84	8	25	6	6.17	5	10
Arteriosclerosis:												
Male	7	121.71	85	190	13	16.23	10	25	5	7.40	5	9
Female	3	74.67	55	105	11	12.27	7	21	3	6.67	5	9
Broncho-pneumonia:												
Male	6	92.00	60	140	14	16.50	10	26	8	7.83	6	9
Female	9	79.44	60	100	17	14.76	9	22	5	7.60	5	11
Lobar pneumonia:												
Male	17	103.59	80	130	63	16.89	10	29	19	8.95	6	14
Female	8	91.00	60	120	23	14.17	9	22	5	6.40	5	9
Portal cirrhosis:												
Male	5	183.00	150	200	10	15.10	10	20	4	7.50	6	9
Female	2	132.50	120	145	1	16.00	----	----	1	8.00	----	----
Hypertrophic cirrhosis:												
Male	----	----	----	----	1	12.00	----	----	----	----	----	----
Female	----	----	----	----	3	11.67	7	15	1	7.00	----	----
Acute nephritis:												
Male	5	98.60	70	130	5	19.80	14	26	3	10.67	8	14
Female	2	115.00	100	130	5	12.40	9	18	1	8.00	----	----
Chronic interstitial nephritis:												
Male	12	97.33	60	140	32	14.84	10	25	8	7.00	5	10
Female	1	120.00	----	----	12	13.25	10	18	1	7.00	----	----
Chronic parenchymatous nephritis:												
Male	3	119.67	90	165	4	14.75	13	18	3	8.33	8	9
Female	7	98.86	60	120	7	15.29	10	21	6	7.83	6	10
Septicæmia:												
Male	18	110.50	80	170	24	19.25	10	31	16	9.13	5	13
Female	8	93.38	60	120	15	18.33	11	30	11	9.45	6	15

TABLE 2.—*Cases and weights of visceral organs of adult Filipinos in certain diseases*—Continued.

Cause of death and sex.	Suprarenals.				Kidneys.											
	Left.				Both.				Right.				Left.			
	Cases.	Weight.			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.
Trauma:		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Male	185	9.08	7	11	556	240.13	160	320	397	117.57	70	170	394	124.14	80	170
Female	52	7.73	5	11	145	218.86	110	320	89	103.62	50	160	89	106.17	50	160
Typhoid fever:																
Male	27	8.59	5	17	128	269.34	210	340	32	134.25	80	190	32	140.75	90	200
Female	12	8.83	5	12	55	258.45	200	300	12	120.58	85	170	12	126.58	90	185
Malaria:																
Male	15	9.07	6	17	44	268.59	190	340	18	136.83	80	190	18	142.17	80	200
Female	1	8.00	------	------	8	286.50	180	340	2	140.00	100	180	2	148.00	96	200
Asiatic cholera:																
Male	------	------	------	------	8	265.38	190	340	------	------	------	------	------	------	------	------
Female	------	------	------	------	5	240.40	200	290	------	------	------	------	------	------	------	------
Amœbic dysentery:																
Male	5	9.20	7	12	42	262.48	185	350	9	136.89	80	190	9	151.00	100	190
Female	4	7.25	4	12	16	245.88	170	340	5	121.00	95	160	5	120.60	80	160
Bacillary dysentery:																
Male	2	8.50	8	9	18	231.50	170	310	4	106.50	90	120	4	113.75	100	140
Female	3	8.00	5	12	14	235.00	170	310	4	98.25	60	130	4	112.50	80	140
Suppurative meningitis:																
Male	8	10.50	8	17	31	261.39	200	340	9	138.22	100	180	9	136.33	90	180
Female	4	11.50	9	18	15	257.53	180	335	8	173.00	120	230	3	151.33	130	180

Tetanus:																
Male	5	9.20	8	10	15	233.80	170	280	15	112.60	90	140	15	121.20	100	150
Female	1	14.00	------	------	4	284.00	180	470	4	132.50	95	210	4	151.50	90	230
Chronic pulmonary tuberculosis:																
Male	41	7.66	4	13	197	249.97	180	316	53	125.66	95	170	53	134.64	100	190
Female	14	8.07	6	12	97	223.71	170	280	20	117.70	95	170	20	124.40	100	180
Tuberculous pneumonia:																
Male	3	7.00	5	10	9	251.78	200	310	4	106.75	80	130	4	118.50	60	150
Female	------	------	------	------	8	249.00	210	295	------	------	------	------	------	------	------	------
Tuberculous meningitis:																
Male	1	8.00	------	------	11	262.82	215	330	1	118.00	------	------	1	117.00	------	------
Female	1	11.00	------	------	7	234.57	200	275	1	119.00	------	------	1	145.00	------	------
General miliary tuberculosis:																
Male	1	7.00	------	------	9	270.44	200	335	2	109.50	97	122	2	112.50	99	126
Female	1	8.00	------	------	4	219.25	150	260	1	124.00	------	------	1	146.00	------	------
Beriberi:																
Male	11	8.73	6	17	34	268.68	200	350	12	131.33	100	160	12	134.83	110	160
Female	4	9.50	9	10	10	252.00	200	300	4	129.25	110	150	4	134.50	110	150
Acute endocarditis:																
Male	1	8.00	------	------	2	268.00	210	326	------	------	------	------	------	------	------	------
Female	------	------	------	------	6	264.00	180	350	------	------	------	------	------	------	------	------
Chronic endocarditis:																
Male	10	9.50	8	11	51	271.92	210	380	15	138.07	95	180	15	150.80	100	190
Female	6	8.33	5	14	39	230.31	165	330	10	105.00	80	150	10	107.00	90	160
Arteriosclerosis:																
Male	5	8.80	6	12	40	259.35	180	350	10	121.90	75	170	10	136.20	105	170
Female	3	8.00	5	12	29	221.59	170	300	6	107.67	90	140	6	114.17	80	160
Broncho-pneumonia:																
Male	3	8.00	7	10	35	274.20	200	380	12	133.58	80	180	12	153.42	95	200
Female	5	8.60	6	12	23	256.09	180	320	15	123.40	90	160	15	139.00	100	180
Lobar pneumonia:																
Male	19	9.53	5	15	97	285.98	200	390	30	149.60	100	200	30	159.73	110	210
Female	5	7.80	5	9	36	273.28	180	340	6	126.67	80	180	6	145.83	80	200

TABLE 2.—*Cases and weights of visceral organs of adult Filipinos in certain diseases*—Continued.

Cause of death and sex.	Suprarenals.				Kidneys.											
	Left.				Both.				Right.				Left.			
	Cases.	Weight.			Cases.	Weight.			Cases.	Weight.			Cases.	Weight.		
		Mean.	Minimum.	Maximum.		Mean.	Minimum.	Maximum.		Mean.	Mini. mum.	Maximum.		Mean.	Minimum.	Maximum.
		g.	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*		*g.*	*g.*	*g.*
Portal cirrhosis:																
Male	4	8.00	7	9	29	265.07	200	350	9	142.56	115	190	9	145.67	110	210
Female	1	8.00			7	229.86	180	320	1	109.00			1	122.00		
Hypertrophic cirrhosis:																
Male					4	241.25	180	290	1	117.00			1	107.00		
Female	1	8.00			3	254.67	220	280	1	147.00			1	117.00		
Acute nephritis:																
Male	3	10.00	6	12	7	303.15	210	380	4	147.00	120	190	4	140.50	70	190
Female	1	10.00			16	284.00	210	390	4	143.75	120	160	4	154.25	130	180
Chronic interstitial nephritis:																
Male	8	8.00	5	12	54	243.28	145	345	10	116.70	70	170	10	117.50	70	170
Female	1	7.00			31	217.97	130	300	6	96.17	60	130	6	101.33	70	140
Chronic parenchymatous nephritis:																
Male	3	7.00	6	9	11	295.55	210	380	4	146.75	90	220	4	128.25	70	210
Female	6	8.33	5	11	17	277.06	200	350	6	156.00	70	240	6	147.50	80	220
Septicæmia:																
Male	16	10.25	5	19	33	289.30	200	370	19	143.42	70	210	19	149.89	70	220
Female	11	10.00	5	15	21	278.62	200	360	12	138.25	100	180	12	144.50	110	200

TABLE 3.—*Cases and percentage increase or decrease of the weights of visceral organs of adult Filipinos in certain diseases.*

[+ Indicates increase, — indicates decrease, from normal.]

Cause of death and sex.	Heart.		Spleen.		Liver.		Pancreas.		Suprarenals.						Kidneys.					
									Both.		Right.		Left.		Both.		Right.		Left.	
Typhoid fever:																				
Male	140	—22	129	+86	137	+12	27	—10	82	—5	27	—3	27	—5	128	+12	82	+14	82	+13
Female	57	—19	56	+110	49	+14	15	+8	33	+0.5	12	+7	12	+14	55	+18	12	+16	12	+19
Malaria:																				
Male	47	—5	45	+244	46	+29	14	+4	20	+7	15	—2	15	—0.1	44	+12	18	+16	18	+15
Female	9	—6	9	+195	7	+36	1	+13	2	—14	1	—29	1	+3	8	+31	2	+35	2	+39
Asiatic cholera:																				
Male	8	—2	8	+22	8	+10	-----	-----	1	—37	-----	-----	-----	-----	8	+11	-----	-----	-----	-----
Female	4	+12	5	—26	4	+9	-----	-----	1	—32	-----	-----	-----	-----	5	+10	-----	-----	-----	-----
Amœbic dysentery:																				
Male	45	—21	43	+1	39	+11	6	+1	23	—16	5	—4	5	+1	42	+9	9	+16	9	+22
Female	18	—17	17	+4	17	+23	3	—10	9	—0.1	4	—26	4	—6	16	+12	5	+17	5	+14
Bacillary dysentery:																				
Male	19	—15	17	—3	18	+8	5	—25	10	—4	2	—34	2	—6	18	—4	4	—9	4	—8
Female	15	—8	15	+44	15	+28	2	—5	8	+7	3	+9	3	+3	14	+7	4	—5	4	+6
Suppurative meningitis:																				
Male	34	—6	27	+37	33	+14	8	—3	21	+10	8	+29	8	+16	31	+9	9	+18	9	+10
Female	19	+13	16	+35	19	+17	4	—1	10	+16	4	+42	4	+49	15	+18	3	+67	3	+42
Tetanus:																				
Male	16	—4	14	—12	13	—6	-----	-----	5	+9	5	—2	5	+1	15	—3	15	—4	15	—2
Female	6	+8	6	—6	5	+8	1	+24	1	+65	1	+42	1	+80	4	+30	4	+28	4	+45
Chronic pulmonary tuberculosis:																				
Male	218	—17	213	+13	220	—5	41	—7	135	—11	41	—12	41	—16	197	+4	53	+7	53	+8
Female	102	—14	92	+16	85	—1	13	—7	55	—12	14	+1	14	+4	97	+2	20	+14	20	+17

TABLE 3.—*Cases and percentage increase or decrease of the weights of visceral organs of adult Filipinos in certain diseases*—Continued.

[+ Indicates increase, — indicates decrease, from normal.]

Cause of death and sex.	Heart.		Spleen.		Liver.		Pancreas.		Suprarenals.						Kidneys.					
									Both.		Right.		Left.		Both.		Right.		Left.	
Tuberculous pneumonia:																				
Male	11	—25	10	+11	8	—4	2	—6	3	—12	3	—16	3	—23	9	+5	4	—9	4	—5
Female	4	—14	3	+5	4	+1	----	----	3	—4	----	----	----	----	3	+14	----	----	----	----
Tuberculous meningitis:																				
Male	13	—18	13	—24	13	—16	1	—33	5	—16	1	—4	1	—12	11	+9	1	+0.4	1	—6
Female	8	—20	8	—27	8	—17	2	+25	7	+1	1	+13	1	+42	7	+7	1	+15	1	+38
General miliary tuberculosis:																				
Male	8	—19	8	+45	8	+10	----	----	7	—9	1	—28	1	—23	9	+13	2	—7	2	—9
Female	3	—30	3	+47	3	+4	1	—19	2	+17	1	—1	1	+3	4	+0.2	1	+20	1	+35
Beriberi:																				
Male	38	+25	36	+114	33	+10	11	+8	19	—7	11	—14	11	—4	34	+12	12	+12	12	+9
Female	10	+33	10	+105	7	+27	3	+38	7	+12	4	+49	4	+23	10	+15	4	+25	4	+27
Acute endocarditis:																				
Male	3	+8	1	+147	2	+19	1	+56	2	—22	1	—28	1	—12	2	+12	----	----	----	----
Female	6	+26	5	+59	5	+28	----	----	3	—4	----	----	----	----	6	+21	----	----	----	----
Chronic endocarditis:																				
Male	52	+46	46	+34	49	+11	10	+8	32	+1	10	+3	10	+5	51	+13	15	+17	15	+21
Female	43	+47	40	+19	41	+10	8	+25	19	—12	6	—13	6	+8	39	+5	10	+1	10	+1
Arteriosclerosis:																				
Male	41	+39	38	—1	35	+10	7	+15	13	+2	5	—11	5	—3	40	—8	10	+4	10	+10
Female	29	+39	29	—4	27	+6	3	—13	11	—16	3	—6	3	+3	29	+1	6	+4	6	+8
Broncho-pneumonia:																				
Male	37	—12	34	+40	36	+8	6	—13	14	+3	3	—12	3	—12	35	+	12	+14	12	+24
Female	25	—1	21	+32	23	+7	9	—8	17	+1	5	+8	5	+11	23	+17	15	+19	15	+31

Lobar pneumonia:																				
Male	99	+3	95	+86	95	+22	17	−2	63	+6	19	+7	19	+5	97	+19	30	+27	30	+29
Female	40	+10	36	+97	39	+20	8	+6	23	−3	5	−9	5	+1	36	+25	6	+22	6	+37
Portal cirrhosis:																				
Male	29	−10	23	+104	26	−15	6	+73	10	−5	4	−10	4	−12	29	+10	9	+21	9	+17
Female	7	+2	6	+121	7	−1	2	+54	1	+10	1	+13	1	+3	7	+5	1	+5	1	+15
Hypertrophic cirrhosis:																				
Male	4	−21	3	+429	5	+29	----	----	1	−25	----	----	----	----	4	+0.5	1	−0.5	1	−14
Female	3	+7	3	+207	3	+5	----	----	3	−20	1	−1	1	+3	3	+16	1	+42	1	+10
Acute nephritis:																				
Male	8	+8	7	+42	7	+0.3	5	−7	5	+24	3	+28	3	+10	7	+26	4	+25	4	+18
Female	18	−1	16	+58	18	+22	2	+33	5	−15	1	+13	1	+29	16	+30	4	+39	4	+45
Chronic interstitial nephritis:																				
Male	56	+21	53	+14	51	+4	12	−8	32	−7	8	−16	8	−12	54	+1	10	−1	10	−5
Female	33	+32	30	+6	33	+2	1	+39	12	−9	1	−1	1	−9	31	−0.4	6	−7	6	−5
Chronic parenchymatous nephritis:																				
Male	13	+3	11	+11	12	−1	3	+13	4	−8	3	−0.2	3	−23	11	+23	4	+25	4	+8
Female	19	+18	16	+40	18	+21	7	+15	7	+5	6	+11	6	+8	17	+27	6	+51	6	+39
Septicæmia:																				
Male	33	−3	33	+77	31	+23	18	+4	24	+21	16	+9	16	+13	33	+20	19	+22	19	+21
Female	27	+1	27	+156	26	+35	8	+8	15	+26	11	+34	11	+29	21	+27	12	+38	12	+36

The weight of the liver is markedly increased in malaria, septicæmia, and lobar pneumonia, and slightly increased in typhoid fever, amœbic dysentery, bacillary dysentery, suppurative meningitis, beriberi, chronic endocarditis, arteriosclerosis, broncho-pneumonia, acute nephritis, chronic interstitial nephritis, and chronic parenchymatous nephritis. The weight is slightly decreased in chronic pulmonary tuberculosis, tuberculous meningitis, portal cirrhosis, and tetanus.

Our data for the pancreas were very meager in the majority of diseases studied. In the few diseases where enough observations were available, it will be noted that the weight of this organ is slightly increased in chronic endocarditis and septicæmia, while it remains almost unchanged in lobar pneumonia. It is slightly decreased in chronic pulmonary tuberculosis. In typhoid fever the weight is slightly decreased in males and slightly increased in females.

The weight of both suprarenal bodies is markedly increased in septicæmia and suppurative meningitis. It remains almost stationary in malaria, chronic endocarditis, arteriosclerosis, and lobar pneumonia. It is slightly decreased in typhoid fever, amœbic dysentery, chronic pulmonary tuberculosis, beriberi, and chronic interstitial nephritis. The weights of the individual suprarenal bodies follow more or less the same tendency as the weight of both suprarenals together.

The weight of both kidneys is markedly increased in acute nephritis, chronic parenchymatous nephritis, septicæmia, and lobar pneumonia. It is only slightly increased in typhoid fever, malaria, amœbic dysentery, suppurative meningitis, beriberi, chronic endocarditis, broncho-pneumonia, and portal cirrhosis. It remains stationary in chronic interstitial nephritis, arteriosclerosis, chronic pulmonary tuberculosis, tuberculous meningitis, bacillary dysentery, and tetanus. The weights of the individual kidneys follow the same tendency of variation as the weight of both kidneys together.

A MITE DISEASE OF TOMATO, TOBACCO, POTATO, AND OTHER PLANTS IN THE PHILIPPINES

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EIGHT PLATES

A mite disease under observation since 1930, but hitherto undescribed in the Philippines, was first noted by the senior writer in the greenhouse of the Bureau of Science to be serious on tomato, tobacco, potato, and other plants. The disease spread so rapidly and its effects were so severe that several thousand tomato and tobacco seedlings and potted potato and other plants had to be discarded. The young leaves of the terminal or axial shoots of the plants affected by this disease show a bronze discoloration and become stiff and narrow. Finally they wilt and dry up from the tip downward, leaving the older expanded leaves unaffected; or they become cupped, crimpled, thickened, or brittle and fail to develop into normal leaves or shoots. The infected plants are stunted, unproductive, and may die prematurely. These infected seedlings and plants show, especially on the underside of the young affected leaves, numerous small, oval, white to pale yellowish green mites, hardly visible to the naked eye.

Since this disease has not been reported before and since its effects are serious, this paper aims to describe it and to report the result of studies made on it thus far in the Philippines. Because of the characteristic dying or thickening, cupping, stunting, and abnormal development of the young leaves of the infected shoots, the name "top crinkle" or "necrosis" is suggested.

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REVIEW OF LITERATURE

Rolfs(9, 10) reported a serious mite disease of tomato in Florida under the name "phytoptosis." He states that first the growing buds and then the fruiting buds of the plant are attacked, whereupon white hairs grow out of the epidermis. From his descriptions and illustrations the mite disease mentioned is different from the Philippine malady and is apparently caused by a different mite species.

Carpenter(2) in 1917 reported a serious mite disease of potato which is prevalent in Hawaii. He states that the shoot of the affected plant dies from the tip downwards, and the young leaves, both terminal and axillary, become bronzed on the lower surface, twist, and curl upon the longer axis, both leaves and shoot becoming abnormally hirsute but soon drying up and dying. He further states that tomato plants growing under similar conditions in Hawaii are also attacked by mites apparently of the same species.

In 1920 Mann, Nagpurkar, and Kulkarni(7) reported a serious "tambera" disease of potato in Poona district, India. On the basis of the symptoms of the infected plants and on their examination of the mites, they believe that the disease is identical with the mite trouble in Hawaii. In 1923 Kulkarni(6) reported a "murda" disease of chili, *Capsicum* spp., stating that the trouble is due to the same mite species as that found on potato in India. Reddick(8) in 1933 found a potato disease in Ithaca, New York, which, apart from minor variations in symptoms, he considered to be identical with the disease of potato in Hawaii(2) and India.(7) He found not only the potato but also the tomato, pepper, and other plants affected.

From the symptoms of mite-infected potato plants described by the above writers, potato plants artificially infected in the greenhouse with mites from the tomato produced symptoms identical with the potato mite disease reported in Hawaii,(2) India,(7) and in Ithaca, New York,(8) and, as will be shown later, this trouble may be caused by the same mite species.

GEOGRAPHIC DISTRIBUTION AND ECONOMIC IMPORTANCE

Because of its seriousness in the greenhouse the disease was surveyed by the senior writer immediately upon its discovery in 1930 and has been under observation ever since. It was found to be commoner in the flower and vegetable gardens in and near Manila than in nearby provinces. In the vicinity of

Manila, tomato, pepper, ampalaya, cosmos, dahlia, and zinnia plants were found commonly affected.

In 1931 a similar disease causing 100 per cent infection on cosmos plants was noted in one of the flower gardens in Los Baños, Laguna Province. In 1932 the senior writer's attention was called to a similar disease on dahlia plants in Singalong and Gagalañgin, Manila.. July 4, 1933, it was found also on cosmos plants and on a weed, *Eclipta alba* (L.) Hassk., from Sañgandaan, Caloocan, Rizal, and August 10, 1933, on pepper seedlings in seed boxes and in plots in San Francisco del Monte, Rizal Province. In November, 1933, a dahlia plant affected with the same disease was noted for the first time at the Trinidad Agricultural School, Baguio, Mountain Province. In January, 1934, tomatoes and potatoes grown in a garden in San Andres, Malate, Manila, were found infested by the same mite. Examination of the leaves of the infected shoots of these plants disclosed numerous mites of the same species as that found in the greenhouse.

Whether the disease is indigenous to the Philippines or was introduced from other countries, it is now common on our economic plants. At present the disease is not widespread, but its becoming established in localities where there are commercial flower and truck gardens may add seriously to our agricultural problems.

SYMPTOMS

Since this disease is reported for the first time in the Philippines and since only slight variations of symptoms are noted on various hosts, a detailed description of these symptoms, especially on tomato, is necessary. The first sign of the disease on the infected shoot of young seedlings or on mature tomato plants is a superficial shiny bronze or brown discoloration on the surface of the succulent stem of the terminal shoot, and, more prominent, on the undersurface of the young leaves (Plate 2, fig. 1). The injury at first is limited to the browning of the epidermal cells (Plate 5), but as the disease progresses the cells of the rapidly growing tissues collapse rapidly and finally die (Plate 2, fig. 2; Plate 4, fig. 1).

Simultaneously with these symptoms the young expanding leaves become narrow, stiff, twisted, or crumpled. They fail to elongate and sooner or later wilt and dry up rapidly as if the top of the plant were scorched by flame (Plate 1, and Plate 2, fig. 2). In some cases these young expanding leaves do not dry up but continue to be narrow, thick, stiff, and crumply as if the infected shoot were suffering from a severe mosaic disease (Plate 3, fig. 4; Plate 4, figs. 2, 3, 4, 6, 7, 8, 9, and 10), while the stem of the plant and the older leaves, although showing bronzed discoloration, remain apparently unaffected (Plate 1; Plate 3, fig. 1). The leaves of the young axillary buds which are expanding at the time of infection also pass through almost the same symptoms as those mentioned above. They also cease to expand, wilt, and dry up quickly (Plate 4, figs. 1 and 2), cr they become narrow, rigid, stiff, thick, and brittle and do not develop into normal leaves (Plate 2, figs. 1 and 2; Plate 4, fig. 4; Plate 5).

As soon as the top is "burned up" the succulent part of the stem of the young plant may be slightly swollen, roughened, or russetted and take on a grayish green color (Plate 3, figs. 1 to 4). Trying to recover, the plant sends out buds from the axis of the blighted leaves or from the normal leaves, but if infestation is heavy, the new buds become infected immediately, and instead of differentiating into typical leaves and stems they become thickened, fleshy, rounded, deformed or atrophied "buds" (Plate 3, figs. 2, 3, and 4; Plate 4, figs. 8, 9, and 10). In plants that are actively growing, several of these atrophied outgrowths are formed and finally crowd each other until they appear as warty protuberances from the axis of the leaves (Plate 4, fig. 9). Plants may continue in this condition for a long time without producing growth, but sooner or later they die (Plate 3, fig. 1). If infestation is light, normal buds may arise from these atrophied "buds," or from the axes of the other leaves. These buds continue to be healthy unless they are again subjected to infestation by mites, in which case the symptoms described above are produced.

Young plants which are infected and subjected to these severe symptoms are stunted and dwarfed and may die before attaining considerable size. Older plants infected in the field or in the greenhouse develop nearly all the above-mentioned symptoms, but excessive production of atrophied warty buds does not occur. These plants do not die, but their productiveness is reduced to a degree depending upon the stage of growth at the time of infection and the number of branches infected. The flowers are also attacked and drop off before they are set. If the young set fruits are attacked, they become hard and russetted and fail to develop. Fruits infested after attaining considerable size may develop to maturity with only blemishes on the skin.

ISOLATION OF THE CAUSAL ORGANISM

Many attempts were made to isolate from the bronzed or infected tips fungi and bacteria by means of the usual isolation methods, but none of these organisms is associated with the disease. In trials where infected tissues were plated without surface sterilization, yellowish bacterial colonies were isolated, but when young colonies were inoculated back by smearing or pricking on the leaves or stem of caged young healthy plants they did not produce the disease.

Experiments were also conducted to determine whether the disease may be due to a virus trouble. Juice from infected plants was injected on the stems or rubbed on the leaves of healthy tomato plants, but the disease has not yet been reproduced artificially. The fact that an infected plant freed from mites will develop new healthy buds, and that such buds will always remain healthy when mites are excluded, suggests that this disease is not a virus trouble. From these limited experiments it appears that the disease is not caused by fungus, bacteria, or virus, but is due to the mites, whose activities are localized and injurious to young leaves and succulent tissues of susceptible host plants.

TAXONOMY AND IDENTITY OF THE CAUSAL MITE

Carpenter(2) did not name the species associated with the mite disease of potato in Hawaii, but stated that it may belong to the same group as the so-called red spider, of the family Tetranychidæ. He further stated that the mite trouble in Hawaii is not the same as that causing phytoptosis of tomato in Florida, caused by *Phytoptus calcladopthora* Nal. Mann et al.(7) described the mite species associated with the serious "tambera" disease of potato in India, but did not attempt to name the mite species. Kulkarni,(6) however, in a later work in 1922 stated that the serious "murda" disease of chili (*Capsicum* spp.) is caused by the same mite species that is associated with the "tambera" disease of potato in India, but does not belong to the red-spider group Tetranychidæ as suggested by Carpenter. He states that it agrees completely with the yellow mite of the genus *Tarsonemus* described by Watt and Mann. Reddick (8) states that the mite associated with the potato disease in Ithaca, New York, is of the genus *Tarsonemus,* but does not give the specific identity of the mites.

In our examinations the mites associated with the Philippine mite disease appeared quite identical with the yellow mite described by Mann et al.(7) and by Kulkarni(6) and belong to the genus *Tarsonemus*.(1)

Egg.—The eggs are round to oval, from 73.0 to 80.3 μ by 87.6 to 102.2 μ long, on the average 75.9 wide and 93.4 μ long (Plate 8, fig. 1). They are transparent to colorless, peculiarly sculptured bodies, firmly attached to the lower surface of the leaves or to the succulent stem. The pattern of the sculpturing is formed in rows of small, opaque, thickened structures on the wall of the egg. Upon hatching the egg breaks at one end, and the egg case persists for a long time on the leaf.

The larvæ.—The larvæ are sluggish, glistening white to slightly colored. They are short oval to long oval, from 65.7 to 87.6 μ wide and from 102.2 to 167.9 μ long, the average 81.7 μ wide and 131.4 μ long (Plate 8, figs. 2 and 3). The young larva has three pairs of legs of almost equal size. The first and second pairs are located anteriorly and the third posteriorly, quite far from the second. The legs are short and composed of five segments terminating in a pulvillus. The body is segmented, soft, and beset with a few hairs.

Adult female.—The adult female is oval, almost twice as long as wide. It is shiny pale yellowish green to light amber, and sparsely beset with short hairs. It is from 87.6 to 116.8 μ wide and 146.0 to 175.2 μ long, the average 100.7 μ wide and 167.9 μ long (Plate 8, figs. 4 and 6). The head is rather prominent, with a pair of stout spines projecting from the dorsal surface. The mandibles are slender, and the palpi are minute and barely visible on the dorsal side. A little below the head is a pair of almost rounded pseudostigmata.

It has four pairs of legs, which are shorter and less developed than those of the male. The first pair is short, stout, and well developed. The second pair is longer than the first, while the third is slender and longest of all. The fourth pair is longer than the first, less developed, slender, delicate, and terminates in two long hairs, one of which is two or more times as long as the other. This pair of legs is dragged.

The tarsus of the first leg possesses a rather large and slightly curved claw and three striated olfactory setæ, the longest located near the middle of the tarsus. The tarsus of the second leg has one olfactory seta at the proximal end, shorter than the longest on the first leg.

Adult male.—The adult male is elongate, about twice as long as wide, and beset with a few hairs. It is more active and much smaller than the female, and measures from 73.0 to 95.5 µ wide and 131.4 to 175.2 µ long, the average 90.5 µ wide and 159.1 µ long (Plate 8, figs. 5 and 7). As in the female, the head is prominent, the mandibles slender, and the palpi minute. The body is widest in the region at which the posterior pair of legs are inserted, and gradually narrows toward the posterior, where a curious anal structure for grasping purposes is found.

The male has four pairs of well-developed legs. The first pair are shorter than the second, the third longest, and the fourth shortest. The first three pairs of legs are provided each with a single pulvillus at the end, while the fourth pair terminates in a short knoblike structure. On each tarsus of the first and second pair is a single olfactory seta. The fourth pair of legs is very peculiar, being more developed and stouter than the other pairs. The penultimate segment is provided with a spur, and near it is found a rather long stout bristle. The tarsus is bent or curved and at about the region of curvature is a long stout hair.

Of the reported plant parasitic species of mite, the Philippine mite belongs to the genus *Tarsonemus,* family Tarsonemidæ,(1) and is similar to, if not the same as, *Tarsonemus translucens* Green, reported on tea in Ceylon and India by Hirst,(4, 5) and the mite disease of potatoes and other plants reported by Carpenter,(2) Mann et al.,(7) Kulkarni,(6) and Reddick.(8) However, until specimens are submitted to specialists for verification, or until further work has been done in this field, for the purpose of this paper the Philippine mite is tentatively referred to as *Tarsonemus translucens* Green. Plate 8, figs. 1 to 7, shows the mite species, *Tarsonemus translucens* Green, which is responsible for the top crinkle or necrosis disease of tomato and other plants in the Philippines.

INFECTION EXPERIMENTS WITH TARSONEMUS TRANSLUCENS AND OTHER GREENHOUSE INSECTS

Methods.—In these infection experiments unless otherwise stated only healthy tomato plants raised under cover with celluloid cylinders from the time the seeds were sown were used. When the seedlings were on their second to third true leaf stage, one plant was transplanted into an 8-inch pot, covered immediately with a celluloid cylinder and then set on a table the legs of which were placed in a shallow pan filled with water.

The insects used in each experiment were taken from plants infected naturally in the greenhouse or in the field or were reared on infected plants for several days before they were transferred onto experimental healthy plants. In transfer the insects were picked one by one with a fine camel's-hair brush, or a portion of the infected leaf on which they were feeding was placed on the experimental plants. In the case of the mite species which are too small to be seen well with the naked eye, a hand lens with 14 to 20 magnification was used to facilitate picking. This method was usually resorted to when transmission was not effected by the placing of infected leaves on which the mites were feeding onto the healthy experimental plants. As controls, healthy plants of the same source and age were used but no insects were transferred. The experiments were kept under observation for twenty-five to thirty days, a period long enough for the development of the disease in question if transmission is successful.

INFECTION EXPERIMENTS WITH TARSONEMUS TRANSLUCENS GREEN

In a series of experiments it was found that *T. translucens* Green can produce the disease if allowed to infest naturally or by artificial means healthy tomato and other plants in the greenhouse. Ten mites from naturally infected plants were carefully picked one by one with a fine camel's-hair brush and placed on the expanded leaves of two caged healthy experimental plants. After eight days bronzing of the leaves and the succulent stem of the terminal shoot was observed on both of these plants. At the end of fifteen days their tops were already "burned up," while the caged healthy check plants which were of the same age and the same source as the experimental plants remained healthy.

In another trial fifteen mites from the above-mentioned infected plants were transferred to one healthy plant, and fifteen mites from naturally infected plants were transferred to each of two caged healthy tomato plants. At the end of six days bronzing was noted on these three plants, and at the end of nine days two of the plants had dead tops. One of the inoculated plants did not develop the "burned up" top until after twelve days. The two check plants, to which no mites had been transferred, were started on the same day, and remained healthy throughout the experiment. Simultaneously with these experiments infected shoots with numerous mites were placed on five caged experimental healthy young tomato plants. These plants also became infected and showed the characteristic symptoms after six to nine days.

September 9, 1930, two young tomato plants were inoculated with five mites each, and two other plants inoculated with ten mites each. After eight days one of the plants started with ten mites showed infection, while the other remained healthy. After thirteen days, however, both plants showed blighted tips. The two plants started with five mites showed no symptoms after eleven days, but after thirteen days one of them showed slight bronzing of the terminal shoot, although burning of the shoot was not noted twenty-five days after inoculation. The other plant was still healthy on this date. In the last series, conducted September 24, 1930, five plants were started with three, five, ten, and fifteen mites, and again successful infection was obtained. The percentage of infection noted was lower and the symptoms were delayed and less severe when fewer mites were used. Table 1 is a summary of results obtained.

Several other observations further confirmed the controlled experiments in the greenhouse. Tomato and tobacco seedlings in seed boxes raised outside of the greenhouse were exposed to natural infection in the greenhouse when they were 3 to 4 inches high. Before exposing the seedlings near the infected plants, the center of the seed boxes, each of which contained twenty-five plants, was covered with a 6-inch-diameter celluloid cylinder, the top of which was covered with muslin cloth. The plants in the other part of the box were left exposed. When the seedlings were examined at the end of fourteen days those covered with cylinders remained healthy, while the uncovered plants showed 20 per cent infection. At the end of twenty-five days, when the final notes were made, those plants which were exposed showed 100 per cent infection, while those inclosed with cylinders were all healthy. Table 1 is a result of one of the series of experiments conducted in the greenhouse.

TABLE 1.—*Number of mites in relation to infection.*

Tomato variety.	Mites per plant.	Plants inoculated.	Plants infected.	Infection.
				Per cent.
Series I, Native Laguna	5	2	1	50
Do	10	2	2	100
Series II, Marglobe	3	5	2	40
Do	5	5	4	80
Do	10	5	5	100
Do	15	5	5	100

In these experiments it was noted that plants, whether subjected to natural or artificial infection, show bronze discoloration within eight to ten days and that the typical "blighted top" stage develops within twelve to fifteen days. When the weather is hot and dry the bronze symptoms are usually developed within five to eight days and "burning up" of the tip appears within ten to fifteen days; this is especially true when a greater number of individuals is used to infest the experimental plants. When the weather is cool, cloudy or rainy, development of symptoms is delayed and typical "burned up" symptoms may not develop even after twenty to twenty-five days. This condition is in conformity with field observations, in that the disease is not usually serious during the rainy season and plants infected may recover and send out new healthy branches. These may become infected sooner or later as the weather becomes favorable, and mites more active.

INFECTION EXPERIMENTS WITH OTHER INSECTS IN THE GREENHOUSE

In a series of experiments, conducted along the lines of those mentioned above, the mealy-bug *Ferrisia virgata* Ckll., thrips (*Thrips* spp.), and red spider have thus far failed to transmit or cause the disease. These insects, therefore, cannot be considered as agents of the disease, even if at times they become numerous in the greenhouse.

CROSS-INFECTION EXPERIMENTS WITH MITES FROM VARIOUS HOSTS

These cross-infection studies, carried on with the method of insect transfer mentioned above, showed that the mite disease of tomato, potato, and dahlia is caused by the same mite species. Mites from tomato, potato, and dahlia, when transferred onto healthy tomato, produce the typical top crinkle or necrosis disease, and when mites from infected tomato are transferred onto dahlia, potato, and other susceptible plants, the typical symptoms are also produced on these plants. Furthermore, on microscopic examination the mites from these hosts appeared to be identical, and no doubt the mite species associated with the disease is *Tarsonemus translucens* Green.

In these experiments the symptoms on the potato began to appear within three or four days. The young leaflets of the terminal shoot or axillary buds show brownish to dark spots, and the underside of the leaves became glossy and finally bronze-colored. The leaves then became somewhat stiff, upright, or twisted. Two or three days after these symptoms were produced, the leaves gradually wilted and soon dried up, the older lower leaves still remaining green (Plate 7, figs. 1 and 2). In addition to these symptoms, the succulent stem showed bronzing, at the base of the petioles lesions were noted, and the leaves were easily detached. In almost all cases the plants died. On dahlia the young leaves also became stiff, narrow, thickened, or the lamina curled downward parallel to the veins in the same manner as observed in the field (Plate 7, fig. 3).

DISSEMINATION IN THE FIELD AND IN THE GREENHOUSE

The rate of spread of the disease in the greenhouse or in the field depends upon the distance between the plants and the prevalence of the mites. In the greenhouse, where mites are quite common, the spread is so rapid that all tomatoes in seed boxes or in pots are infected, develop "burned" shoots, and die in the course of a few days. In the field the initial infection usually comes from other infected host plants or from infected seedlings which are transplanted. Field dissemination is usually negligible when plantings are far apart, but when they are close together and touching one another, as in the seed boxes and seed beds, dissemination is very rapid.

December 1, 1930, tomato seedlings that appeared to be healthy in the greenhouse were planted about 70 cm apart at the pathological plot in Manila. This plot was newly opened and far from home and flower gardens known to be infected with the disease. December 20, diseased plants were noted at different regions of the field, and since no other plants nearby were affected the source of infection no doubt came with the tomato seedlings, which must have had mites at the time of planting but had not developed the symptoms. Since the diseased plants were found at different regions of the field, notes were made on the rate of spread of the disease. January 20, 1931, when the final notes were made, a slight increase of infection on the row was noted but the total increase of diseased plants in the field was negligible. Only the plants near the diseased plants became infected, indicating that the spread of the disease from plant to plant in the row or between rows is negligible. Table 2 contains the summary of results obtained.

Whether this disease will ever become a serious pest in the field on tomato or other economic plants in the Philippines, remains to be seen. In Hawaii and in India an identical disease has been reported widespread and very serious on potato and on chile (*Capsicum* spp.). In the Philippines it is as yet not widespread or serious, but there is great danger of its spreading further and becoming established in localities where commercial planting of tomatoes, potatoes, tobacco, and other susceptible crops is practised.

TABLE 2.—*Rate of spread of the disease in the pathological plot in Manila during the 1930 season.*

Variety.	Number of plants.		
	Observed.	Infected.	
		Dec. 20, 1930.	Jan. 31, 1931.
AMERICAN VARIETIES.			
1. Burpee's Dwarf Giant	17	4	6
2. Dwarf Stone	17	1	1
3. Earliana	32	2	8
4. Greater Baltimore	15	3	5
5. John Baer	16	0	0
6. Marglobe	16	2	8
7. Marglobe (Stokes)	17	0	2
8. Matchless B. P. I.	28	4	4
9. Maule's Columbia Wilt Proof	17	3	8
10. Maule's Success	16	2	3
11. Norton Wilt Resistant	15	1	7
12. Ponderosa	15	2	4
13. Ponderosa, Giant	17	1	1
14. Ponderosa, Yellow	17	1	1
15. Perfection	28	1	2
NATIVE VARIETIES.			
19. Laguna	17	1	1
27. Tarlac	10	0	0
25. San Isidro (1)	17	3	8
26. San Isidro (2)	33	1	1
16. Calumpit 1	37	1	1
17. Calumpit 3	14	0	0
18. Calumpit 4	74	3	10
24. San Carlos, Pangasinan (1)	14	2	4
24. San Carlos, Pangasinan (2)	17	1	2

RELATIVE SUSCEPTIBILITY OF VARIOUS TOMATO VARIETIES

As far as can be determined, many American and native tomato varieties are susceptible to top crinkle or necrosis. In these trials, 35-day-old tomato seedlings of each variety are planted in 8-inch pots and exposed to natural infection in the greenhouse for thirty days. Among the American varieties tested which were found susceptible are Burpee's Dwarf Giant, Dwarf Stone, Earliana, Greater Baltimore, John Baer, Marglobe, Matchless, Maule's Columbia Wilt Proof, Maule's Success, Norton Wilt Resistant, Ponderosa, Giant Ponderosa, Yellow Ponderosa, Perfection, Sunnybrook Earliana, Red Pear, Yellow Pear, Burpee's Matchless, Golden Dwarf Champion, Red Cherry, Burpee's Self Pruning, Sparks Earliana, Acme, True Giant Ponderosa, Stone, The Burpee, Yellow Plum, June Pink, Trucker's Favorite, Early Detroit, Bonny Best, Whole Salad, Penn. State Earliana, Livingstone Globe, Gulf State Market, Beefsteak and Burpee's, Fordhook First. Among the native varieties infected are those from Muñoz, Calumpit (Selections 1, 2, 3, 4), Pangasinan (Selections 1 and 2), Batangas, Tarlac (Selections 1 and 2), and the Lemery Wild Cherry tomato. The results, therefore, indicate that many of the common varieties grown in the United States and in the Philippines are susceptible, and none of the varieties show a high degree of resistance to the disease.

HOST RANGE

From available literature, *Tarsonemus translucens* Green has been reported attacking tea in Ceylon and India.(4, 5) A mite species of *Tarsonemus*, probably *T. translucens*, has been reported on potato, and tomato in Hawaii by Carpenter;(2) on potato and "guvar" *Cyamopsis psoralioides* in India by Mann, Nagpurkar, and Kulkarni;(7) on chili (*Capsicum*), zinnia, dahlia, tagetes, *Mirabilis jalapa,* Cape gooseberry, *Amaranthus polygonus* and *Physalis minima* in India by Kulkarni;(6) on a number of domestic varieties of potatoes, *Physalis* spp., *Nicandra physaloides, Datura stramonium, Capsicum* spp., and tomato in Ithaca, New York, by Reddick.(8)

In the Philippines, tomato, potato, pepper, "ampalaya," dahlia, cosmos, zinnia, and a weed plant, *Eclipta alba* (Linn.) Hassk., are naturally affected. In a series of inoculation experiments in the greenhouse the above-mentioned hosts, and tobacco, cowpea, "mungo," "sitao," papaya, pechay, Chinese cabbage, radish, mustard, and mango seedlings are equally susceptible to the mite trouble, while sincamas, rice, onion, and corn appeared to be more resistant. The result shows that many of our economic plants are susceptible. Since the scope of this work is limited, other host plants may eventually be added to the list.

It is interesting to note that the symptoms produced vary with the different hosts. In general, however, the characteristic bronzing, stiffening, thickening, narrowing, and curling of the young leaves of the shoot and dying of the tip occur. In susceptible plants with wide leaves, such as tobacco, dahlia, pechay, radish, etc., the blade may be severely cupped laterally, thickened, brittle, and much reduced in size (Plate 7). In some hosts, such as cosmos, the leaves as a result of the disease become so reduced in size, thickened, and deformed, as to appear filiform. They are bunched up or crowded on the infected shoot which in turn fails to elongate normally (Pate 7, fig. 4).

CONTROL MEASURES

Rolfs(9) in his studies on the tomato mite disease in Florida found flowers of sulphur sprinkled on the plants as efficacious as sulphur spray. When "phytoptosis" was the only disease to be treated, he found that flowers of sulphur was more economical. Carpenter(2) recommends spraying with lime-sulphur as an effective means of controlling the potato-mite disease in Hawaii. In India Mann et al.(7) showed that spraying with lime-sulphur wash or dusting with sulphur, giving all together three dressings, beginning at the time when the potato plants are three weeks old, produced excellent results in the field. Kulkarni(6) also recommends lime-sulphur as an effective control in the field for the "murda" disease of chili. Reddick(8) suggested the use of naphthalene for the fumigation of mites in the greenhouse.

Our control experiments in the greenhouse with infected dahlia and tomato plants showed that the disease can be checked quite effectively by spraying with an aqueous solution of 1 : 1,000 or 2 : 1,000 nicotine sulphate to which 5 grams of white Chinese soap have been added. Tests with an aqueous solution of 2 : 1,000; 3 : 1,000, or 4 : 1,000 nicotine sulphate alone did not give satisfactory results. Soap solutions with concentrations of from 0.4 to 0.6 per cent gave fairly good results but are more effective when nicotine sulphate has been added. These soap solutions, therefore, are recommended in places where nicotine sulphate cannot be obtained.

Lime sulphur (33° Baumé) diluted with water in proportion of 1 : 75 and 1 : 100 was found very effective and more satisfactory than the nicotine sulphate-soap solution combination. Dilutions 1 : 25 and 1 : 50 were also tried and proved very effective but they cause serious burning of the leaves. Good results were also obtained by spraying with the Sherwin Williams dry lime-sulphur made into 0.5, 0.75, and 1 per cent solutions or by dusting with the dry lime-sulphur while the plants are not moist or wet. Since very effective control was obtained with lime-sulphur (33° Baumé) diluted to 1 : 75 and 1 : 100 and with Sherwin Williams dry lime-sulphur, these solutions or the dry lime-sulphur dust should be also used. Plate 8, fig. 10, shows the recovery of an infected tomato plant which has been sprayed with 1 : 100 lime-sulphur, and a dahlia plant sprayed with an aqueous solution of 2 : 1,000 nicotine sulphate to which 5 grams of soap were added.

Success was also obtained by fumigation of the infected plants with "cyanogas" at the rate of about one teaspoonful or more of the powder to about a cubic meter of space. Naphthalene was recommended by Hartzell and Wilcoxon(3) for certain greenhouse insects. In fumigating the plants should be placed in air-tight chambers or covered with canvas for five to fifteen minutes.

The success in the control of this mite, obtained either by spraying, by dusting, or by fumigating, largely depends on the thoroughness and frequency of application. In spraying, although not so very necessary, more effective results can be obtained by directing the solution to the underside of the leaves as the mites are more numerous there. Rogueing and burning of infected plants, cleanliness in the garden or field and isolation of sprayed or fumigated potted plants to avoid reinfestation, are precautions to be observed in order to minimize the spreading of the disease.

SUMMARY

1. A mite disease serious in the greenhouse on tomato and other economic plants is reported for the first time in the Philippines.

2. Because of the characteristic drying up or cupping and crumpling of the young leaves of the shoots and of the axillary buds, the name "top crinkle" or "necrosis" is suggested.

3. The disease is still limited in its distribution in the Philippines, having been found in Manila, Los Baños, Laguna, and in the Trinidad Agricultural School, Baguio, Mountain Province, naturally affecting tomato, pepper, ampalaya, dahlia, cosmos, zinnia, and a weed plant, *Eclipta alba* (L.) Hassk.

4. From available literature the Philippine mite disease appears to be caused by the mite species that is serious on potato, chili (*Capsicum* spp.), and other plants reported in Hawaii, India, and New York.

5. The symptoms of the disease on tomato are described in detail. Crinkling or "burning up" of the top of young shoots are characteristic symptoms of the disease.

6. Attempts to isolate a fungus or bacterium from the infected areas showed that the malady is not due to any of these.

7. Controlled cage experiments in the greenhouse showed that the disease is caused by a small yellowish green mite, which in all morphological characteristics is similar to, if not the same as, the yellow mite on tea, *Tarsonemus translucens* Green, described in Ceylon and India. The Philippine mite species is tentatively identified as *Tarsonemus translucens* Green.

8. A species of a mealy-bug, a thrips, and a red spider, which are very common greenhouse insects, failed to produce the disease.

9. The plants which became infected naturally or artificially by mites in the greenhouse showed the bronze symptoms within eight to ten days, and the "burned up top stage" is usually developed within twelve to fifteen days. A larger number of mites transferred to each plant, under favorable condition, hastens the appearance of these symptoms.

10. Cross-infection experiments in the greenhouse and microscopical examination of the causal mites showed that the disease of tomato, potato, pepper, dahlia, and other plants in the Philippines is possibly due to the same mite species, *T. translucens* Green.

11. The dissemination of the disease in the field is slow when the plants are far apart; but in the greenhouse, especially in seed boxes and in beds where plants are sown closer together, the spread is very rapid.

12. Tests on the susceptibility of the different American and Philippine native tomatoes indicated that all of them are susceptible to the disease.

13. Artificial and natural infection showed that the mite affects a number of economic plants. The tomato, tobacco, potato, pepper, eggplant, pechay, mustard, cowpea, mungo, sitao, ampalaya, papaya, mango, dahlia, cosmos, zinnia, and a weed plant, *Eclipta alba* (Linn.) Hassk., are susceptible to the disease and may serve as host plants for the mite. Sincamas, rice, and corn were found not seriously affected.

14. Experiments on control showed that spraying infected plants with 1 : 1,000 or 2 : 1,000 nicotine sulphate plus 5 grams Chinese laundry soap; or spraying with lime-sulphur (33° Baumé) diluted 1 : 75 or 1 : 100; or 0.50, 0.75, and 1 per cent solution of Sherwin Williams dry lime-sulphur; or dusting plants with dry lime-sulphur, gave very good results. Fumigating them with "cyanogas" also gave satisfactory results.

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ILLUSTRATIONS

PLATE 1

Native tomato seedling affected with the top blight, or necrosis disease. Note the "dried up" young expanding leaves of the terminal shoot, leaving the top as if scorched by flame. The lower older leaves remain unaffected.

PLATE 2

Young tomato plants naturally infected in the greenhouse, showing different stages of the disease.

FIG. 1. Plant showing the early but marked bronzing or browning of the young leaves and of the succulent part of the shoot. Note also that the youngest leaflets are somewhat narrow, stiff, and rather upright.

2. The same plant shown in fig. 1, but now it has developed more advance symptoms of the disease. The leaves of the terminal shoot are now wilted and dried up. (Five days older.)

PLATE 3

A "wild" tomato plant (Cherry variety) naturally infected with top blight in the greenhouse, showing the effect of the disease on the flowers, young fruits, and terminal shoots.

FIG. 1. Infected terminal shoot showing symptoms on young fruits and leaves.

2. Enlarged picture of the infected shoot shown in fig. 1. Note the typical russetting or cracking of the skin of the fruit. These fruits became hard, failed to enlarge, and finally dropped off.

3. Infected shoot showing the russetting and swelling of the succulent stem. Killing of the terminal bud, and the dropping off of the flowers before they are set are also characteristic symptoms of the disease.

4. A shoot of an infected plant, showing the various malformations and the atrophied leaflets of the infected terminal shoot. Note that the leaflets are much thickened, crumply, and greatly reduced in size.

PLATE 4

FIGS. 1 to 4. Tomato plants naturally infected in the field. Earliana variety showing severe symptoms. The leaflets narrowed, thickened, crumpled, and failed to expand normally. The tip of the shoot wilted and dried up. The compound leaf on the extreme right also shows severe symptoms on the lower young leaflets.

5 to 8. Greater Baltimore variety showing symptoms almost identical with those in figs. 1 to 4. Note the stunted growing tip of the shoot. The compound leaf on the extreme left is healthy, taken just below the infected leaves.

FIG. 9. Shoot of infected young seedling in the greenhouse showing crowded and stunted buds from the axes of infected and healthy leaves. The leaves were removed in order to make more visible the deformed or atrophied buds.

10. Terminal shoot of the infected seedling in the greenhouse showing the atrophied leaflets and petioles of the young compound leaves of the terminal shoot. Note also that the axillary buds are stiffened, thickened, and crumpled due to infection.

11. Compound leaf from the seedling illustrated in fig. 10 taken from the lower part of the stem.

PLATE 5

Microphotograph showing the extent of injury of an infected young stem.

FIG. 1. Cross section made from the region just a few centimeters below the tip of the shoot.

2. An enlarged section from fig. 1. Note that the browning of tissues is at first limited only to the epidermal layer. In very young and rapidly growing tissues collapse of the cells takes place rapidly.

PLATE 6

Photographs showing symptoms of top crinkle or necrosis on tobacco seedlings.

FIGS. 1, 2, and 5. Infected plants with typical bronzing and cupping symptoms. Note the lateral cupping of the leaves and drying of the young expanding leaves of the terminal shoot. The plants are stunted and failed to grow normally.

3 and 4. Healthy plants.

PLATE 7

Photographs showing the symptoms of top crinkle or necrosis on potato plants naturally infected in the greenhouse.

FIG. 1. Healthy leaves.

2. Infected leaves showing the typical symptoms. These plants developed typical bronzing of the stems and leaves, which later became stiff and crumply, and finally wilted and dried up. The "drying up" of the leaves generally starts from the top downward.

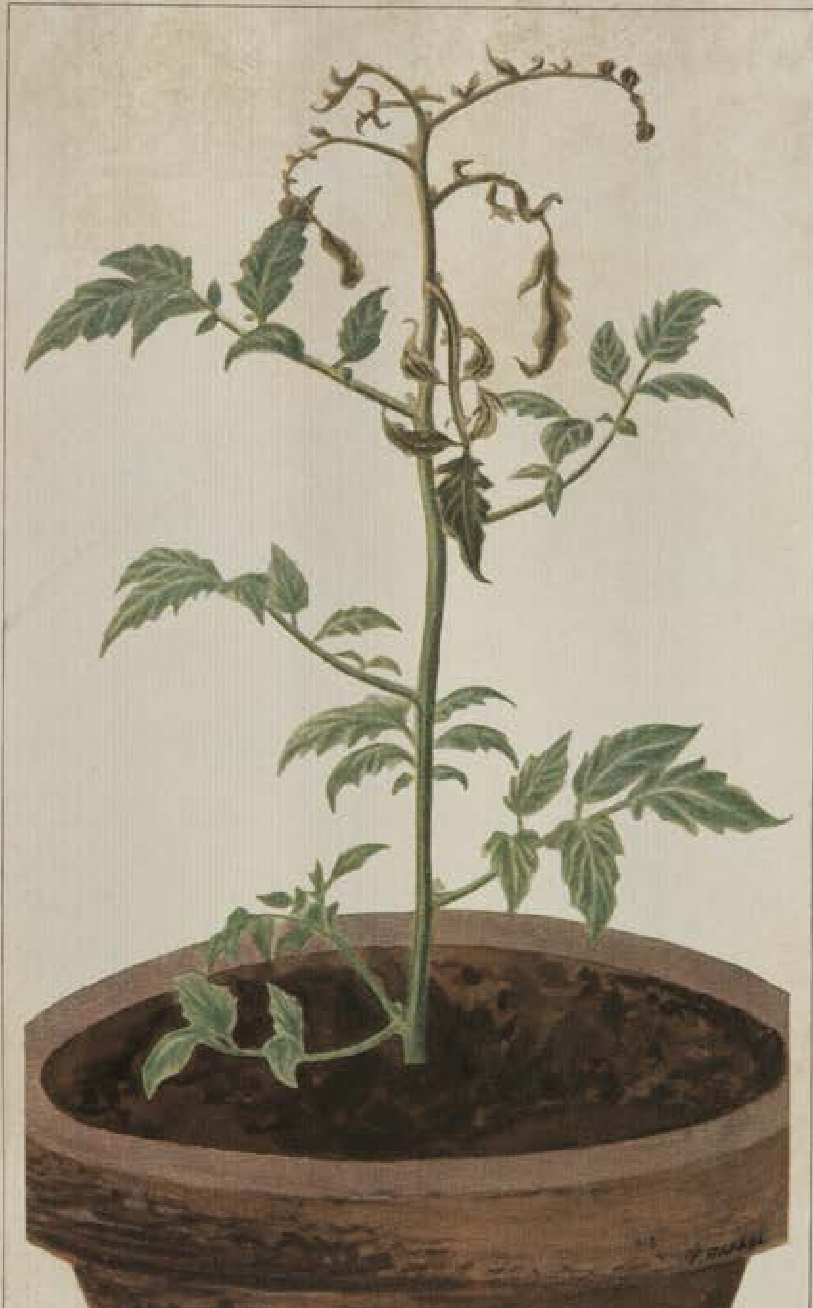
3. Photograph of one of the shoots of an infected dahlia plant from Mrs. de Santos's flower garden showing the severe lateral curling, twisting, or cupping of the young leaves. These leaves are thickened, stiff, narrow, and very brittle. They failed to develop normally. Note the older lower leaves which remain unaffected.

4. A shoot of an infected cosmos plant from Sañgandaan, Caloocan, Rizal Province, showing the typical symptoms. Note the atrophied, thickened leaves and excessive bud development on the shoot. As a result of infection the shoot also failed to elongate, thus acquiring a "bunched up" or crowded appearance.

PLATE 8

The tarsonemid mite and sprayed and unsprayed dahlia and tomato plants.

FIG. 1. Egg.
2. Larva, dorsal view.
3. Larva, ventral view.
4. Adult female, dorsal view.
5. Adult male, dorsal view.
6. Adult female, ventral view.
7. Adult male, ventral view.
8. Potted infected dahlia plant from a flower garden. This plant was sprayed with an aqueous solution of 2:1,000 nicotine-sulphate to which 5 grams of soap were added. The other infected shoots were removed.
9. Infected tomato plant showing dried tops. The axial buds which developed also became infected and are now badly crumpled.
10. Infected tomato sprayed once with lime-sulphur (33° Baumé) diluted 1 : 100. At X is the terminal shoot which was "burned up" twenty-two days ago. The axial buds which developed are all healthy, showing complete recovery from disease (twenty-two days after treatment).
11. The same dahlia shoot as in fig. 8 showing complete recovery and development of a new healthy shoot with normal leaves. Note the infected leaves at the lower part of the stem before treatment.



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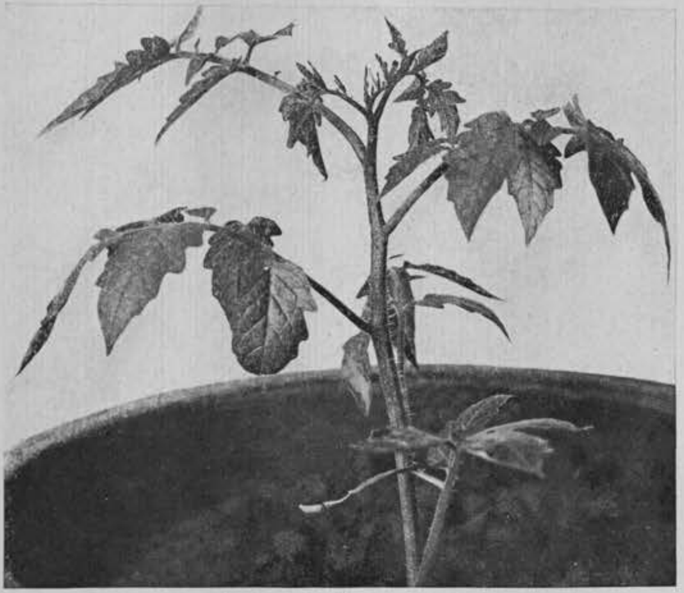

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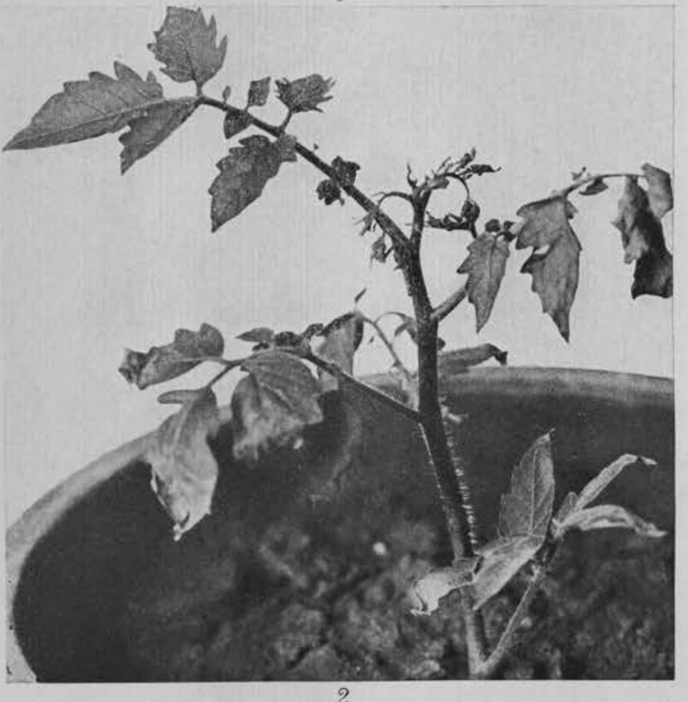

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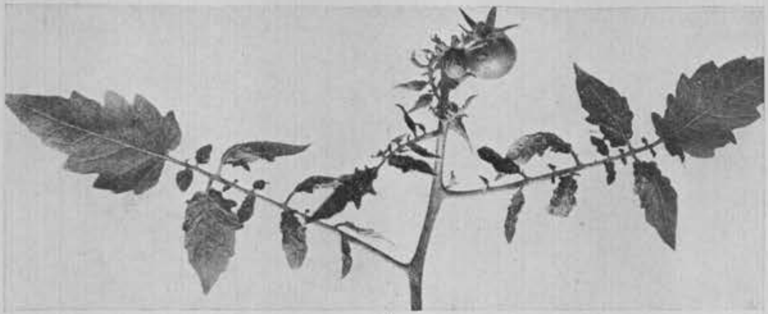

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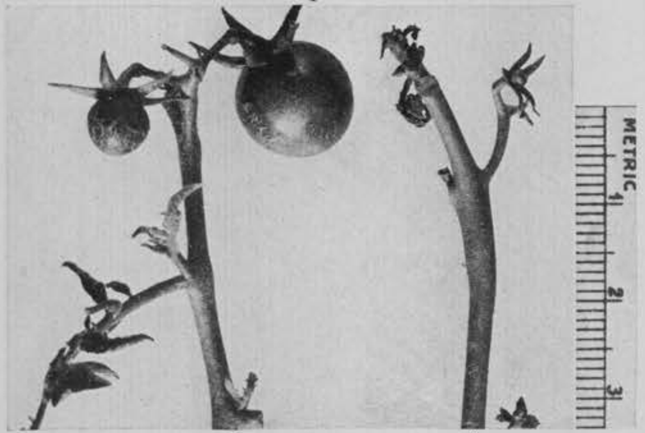

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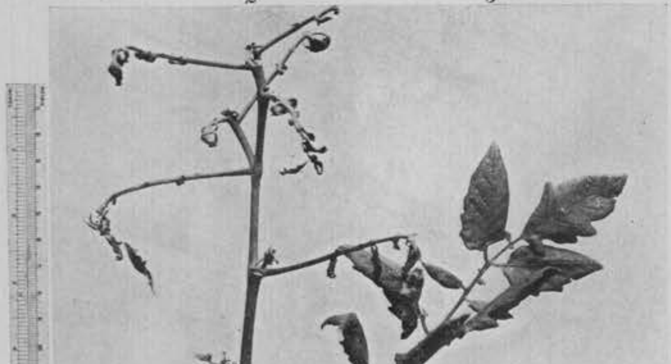

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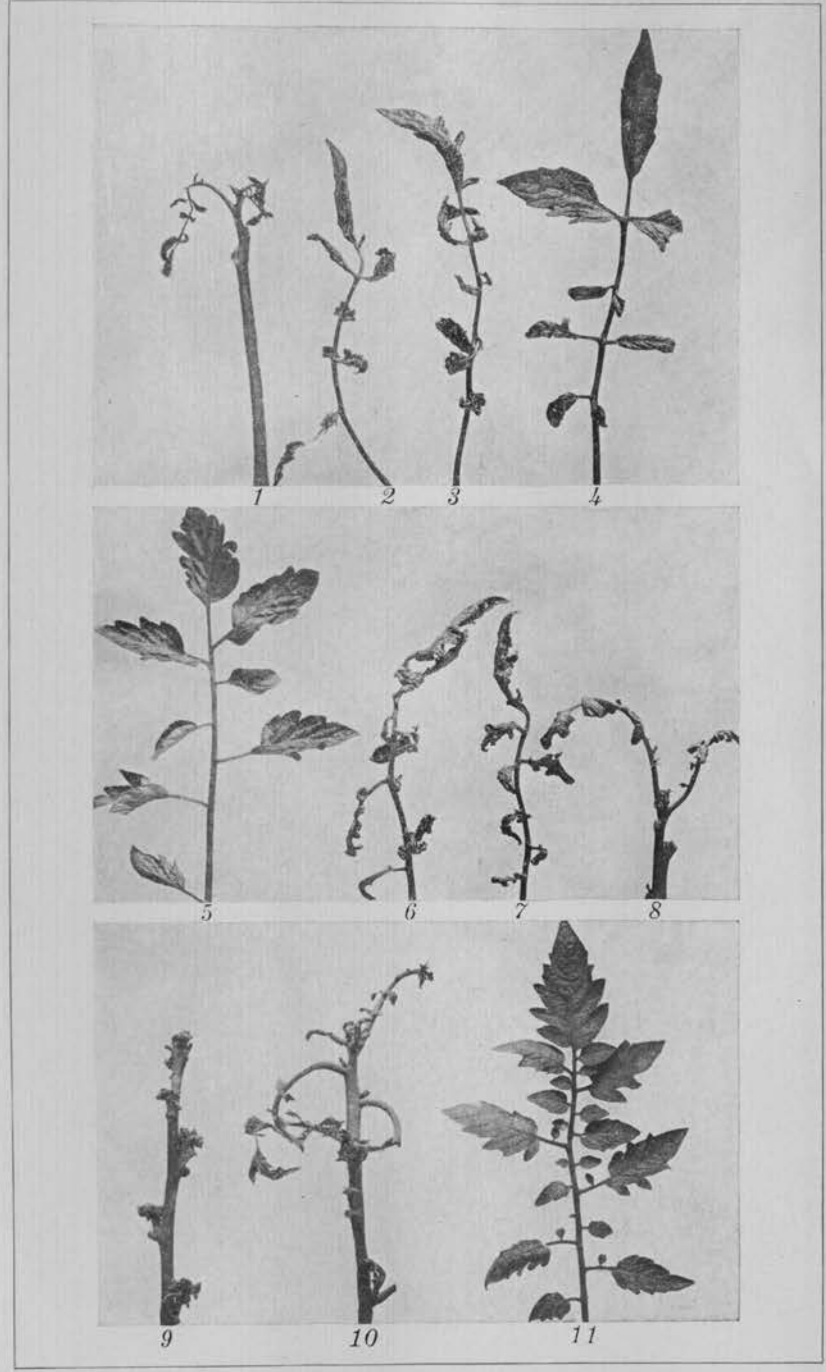

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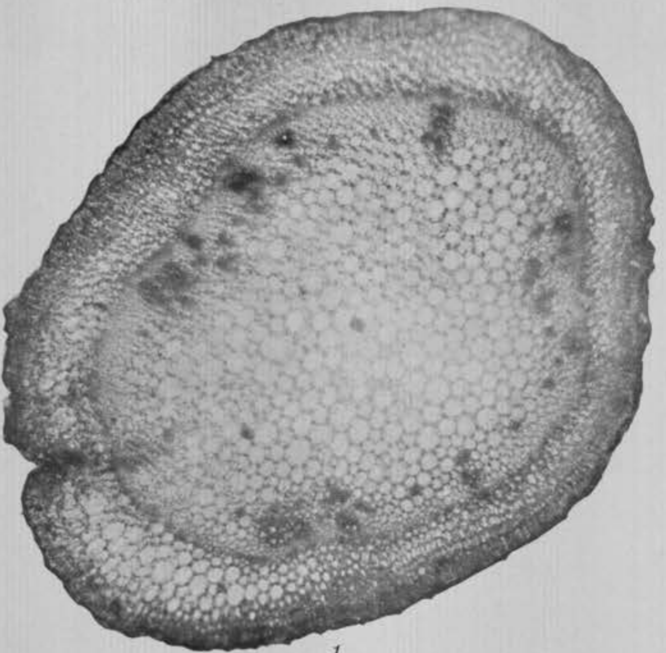

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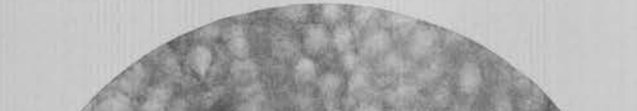

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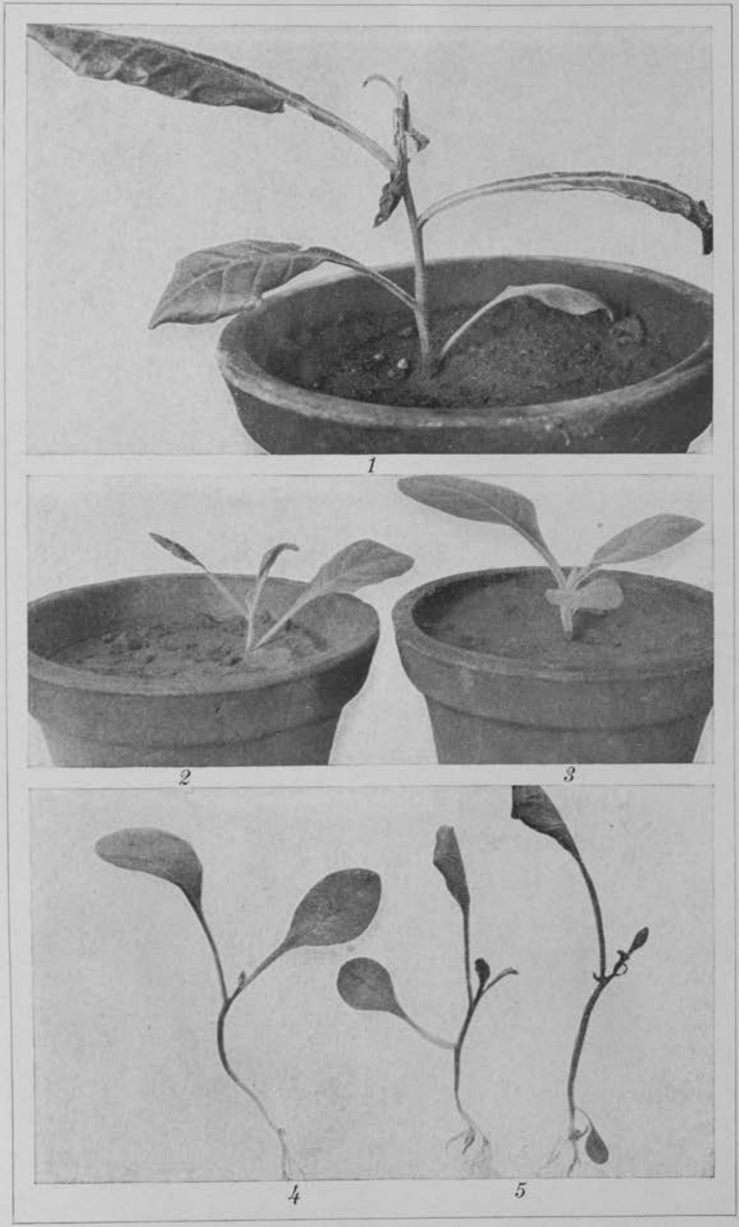

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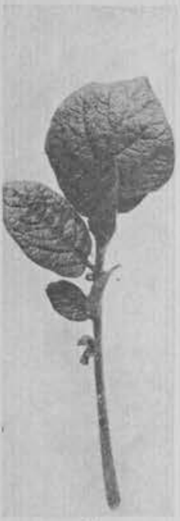

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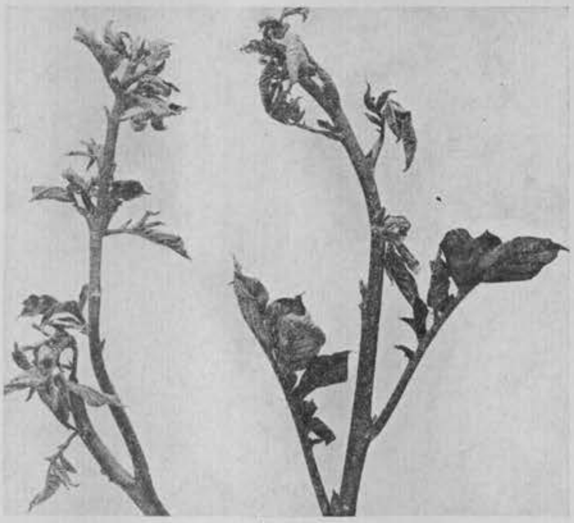

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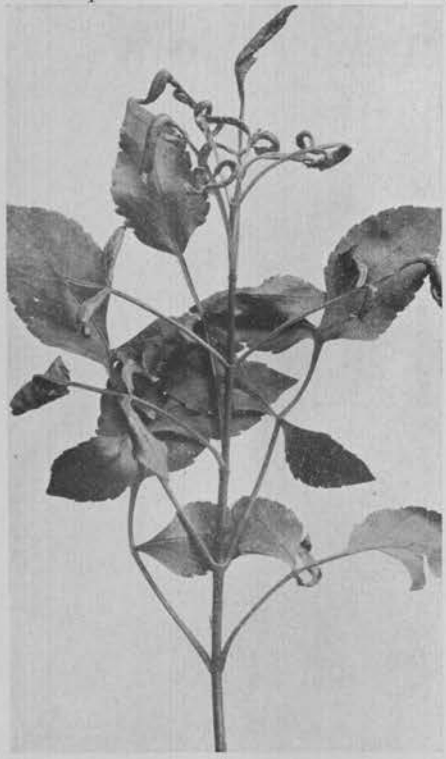

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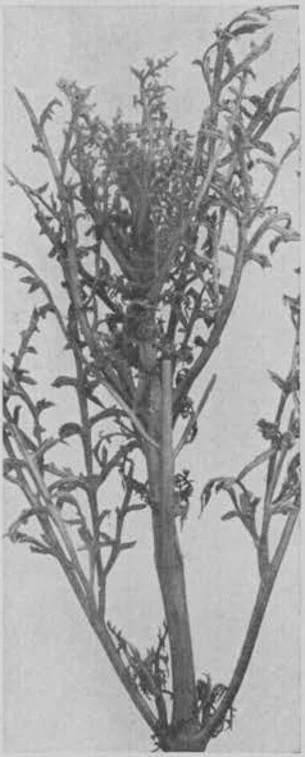

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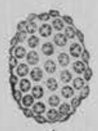
PLATE 7.



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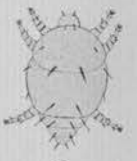

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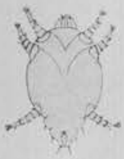

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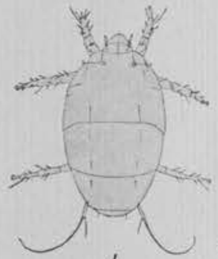

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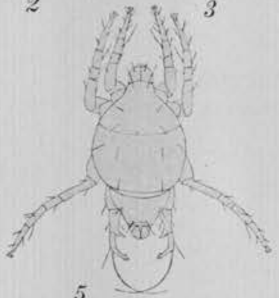

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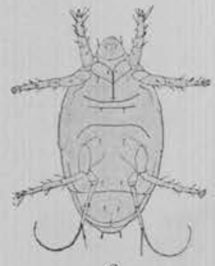

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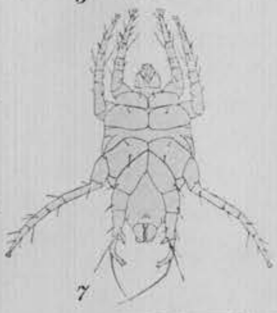

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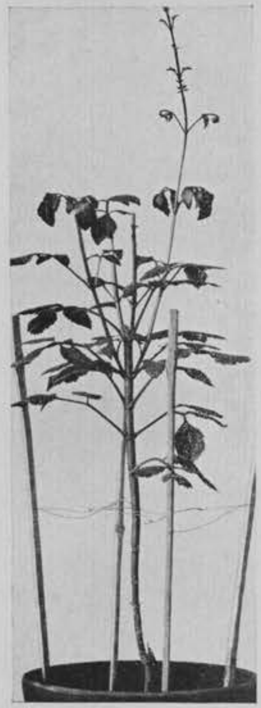

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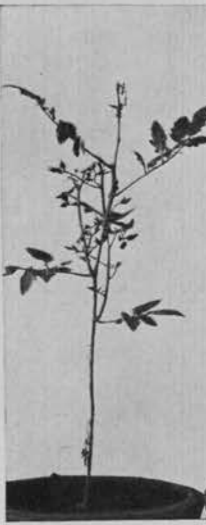

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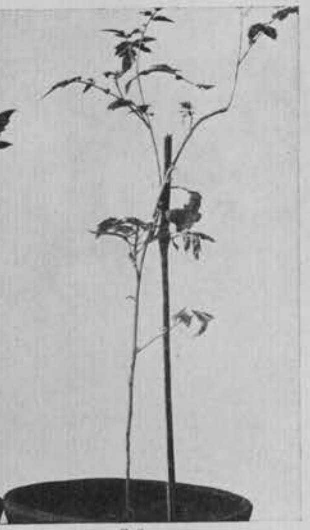

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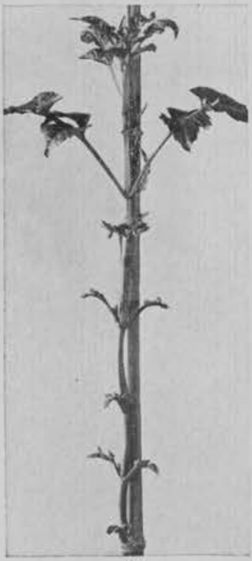

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PLATE 8.

KANDULI FISHERIES OF LAGUNA DE BAY PHILIPPINE ISLANDS

REMEDIAL AND REGULATORY MEASURES FOR THEIR REHABILITATION[1]

By Deogracias V. Villadolid

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The most conservative estimate of the value of the *kanduli*[2] fisheries of Laguna de Bay, made in 1928, placed it at about 1,000,000 pesos a year; at present (1934), it is doubtful if the annual harvest is worth one-half that sum. The fall in yield may be traced to the apparent depletion of the fisheries in question, due principally to the indiscriminate catching of the fish by the *pukot* (drag seine).

FACTORS OF DEPLETION

On the basis of more than ten years' observation, the following may be mentioned as important factors of depletion:

1. The pukot.—The pukot (drag seine) withdraws the bulk of fish from Laguna de Bay, and therefore is very destructive to the fisheries, especially to the kanduli, the spawning and brooding seasons of which coincide with the best fishing season of the pukot. Laguna de Bay is too shallow a body of water, being only from 3 to 6 meters deep in the open, to withstand the severe effect of the pukot, the thorough working gear of which does not allow the fish in the lake much chance of escape. Moreover, the operation of this drag seine disturbs the ecology of the bottom of the lake, destroying both plants and animals growing there. These demersal forms are the principal source of food for the food fish, especially the kanduli, which is a bottom-feeder.

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[1] Contribution No. 10 from the Fish and Game Administration, Department of Agriculture and Commerce.

[2] The kanduli of Laguna de Bay belongs to the catfish group, *Arius* species, family Ariidæ.

Our studies on the composition of the catch of the important commercial fishing gear in Laguna de Bay, operated during 1931 and 1932, showed that pukot withdrew about 99 per cent of the total catch of kanduli; about 94 per cent of the *biyang-puti, Glossogobius giurus* (Buchanan-Hamilton); about 97 per cent of the *ayun̄gin, Therapon plumbeus* (Kner); and about 99 per cent of the *dalag, Ophicephalus striatus* Bloch, taken from the lake during that period.

Increase in the number of fishermen and greater effort naturally tend to deplete a given fishery. The Bureau of Commerce and Industry of the Philippine Islands is authority for the following figures: In 1918, 177 fishing establishments were catching fish in Laguna de Bay. By 1925 the number of such establishments was 1,045, an increase of 868, or an average increase of 124 a year. Within the last five or six years, moreover, the pukot fishermen of Laguna de Bay began to employ motor boats in operating their drag seines, which markedly accelerated the coverage of fishing areas.

2. The dominance of young and immature kanduli in the local markets.—Large quantities of immature fish are sold in the local markets. This is especially true of the kanduli, the major bulk in the fish baskets being composed of *tiguiti* (Tagalog name for small, young, and immature kanduli).

3. Large market demand for prawns, snails, and small fish.—There is a large demand for prawns, snails, and small fish, all of which serve as food for commercial fish. This demand naturally gives rise to ruthless methods of catching these animals. Depletion of the food of fish naturally will tend to decrease the number of fish. The duck industry in Laguna de Bay regions, which requires a large amount of protein food, is extensive. In has been found in the College of Agriculture that the best protein food for chickens, swine, and other domestic animals is prawns and snails. The demand for this protein feed in the Laguna de Bay regions is so large that local sources alone cannot meet it, and a considerable portion has to be imported.

4. Periodic occurrence of masamang tubig (bad water) in Laguna de Bay.—It was observed that periodic occurrence of *masamang tubig* is responsible for the wholesale death of fish and other animals in localities through which the bad water passes. It is believed that this water, which occurs at certain times of the year, originates from the salt water of Manila Bay. This water is further polluted with decaying algæ (locally known as *lia*). Chemical examinations of this water in the laboratory of agricultural chemistry, College of Agriculture, showed that it is almost devoid of oxygen, and highly charged with poisonous gases. Its chlorine content is much higher than that of the ordinary lake water. This bad water is a factor of depletion since it kills fish. Even the hardy snails and other mollusks succumb to its effect.

POSITIVE SIGNS OF DEPLETION

The following are some of the positive signs of the depletion of kanduli in Laguna de Bay.

1. Increased percentage of young in the catch from 1927 to 1932 per unit of effort or gear.—The percentage of young in the catch is a useful indicator of the condition of the fisheries. An increase in the percentage of young in the catch is a sign that the fish are getting scarcer. In 1927 and 1928 a series of determinations at weekly intervals was made to ascertain the composition of the catch of two nonselective gears which catch kanduli on a commercial scale. The average monthly catch of *baklad-laot* or *baklad-alañgan* was 60 per cent immature kanduli, and 50 per cent immature kanduli for the pukot. (In the case of the pukot, 50 per cent of the immature kanduli contained in the catch did not include the countless eggs, embryos, and fry that were destroyed during the spawning and brooding season.) Our 1931–1932 determinations showed that the pukot had a monthly average catch of 60 per cent of immature kanduli; the baklad had 63 per cent. The foregoing determinations were based upon the sampling method. Fortunately we also have records of similar determinations of the catch of the baklad, but based upon treatment of the entire catch. This was made possible by the establishment of two fish corrals (baklad) under the management of the College of Agriculture limnological station, where we had control over the disposal of the catch. Because of the completeness of these data, we think that the results therefrom are more representative than those obtained from the two methods of determination. We found that they agree fairly well as to the percentage of young in the catch, as recorded in Table 1.

TABLE 1.—*Percentage of immature kanduli. Monthly average from the year's determinations.*

Period.	Immature kanduli.	
	By sampling.	By treatment of entire catch.
	Per cent.	*Per cent.*
1927–1928	60	
1931–1932	63	
1931		59.3
1932		69.2

2. Gradual reduction of kanduli in the catch, and its replacement by biyang-puti.—Another significant indication of the depletion of the kanduli fishery is the gradual replacement of kanduli in the catch by biyang-puti. About five years ago the bulk of the catch of baklad in Laguna de Bay was made up of kanduli, biyang-puti forming only a minor portion. From 1931 to 1932, however, biyang-puti began to dominate the catch. The kanduli gradually decreased until it represents only a minor portion, while the biyang-puti forms the dominant constituent of the catch of the fish corrals. The record of the total catch of the two baklad run by the College of Agriculture limnological station for 1931 and 1932 shows the ratio between the kanduli and the biyang-puti found in the catch. Table 2 presents the monthly average taken from records of the daily catch.

TABLE 2.—*Monthly catch of two baklad, showing the relation between kanduli and biyang-puti during 1931 and 1932.*

Month.	1931		1932	
	Kanduli.	Biyang-puti.	Kanduli.	Biyang-puti.
January			1	3.09
February			1	0.66
March	1	1.89	1	1.53
April	1	2.06	1	2.93
May	1	0.86	1	3.36
June	1	1.24	1	2.33
July	1	0.88	1	1.50
August	1	1.88	1	1.50
September	1	0.46	1	1.21
October	1	0.42	1	2.78
November	1	1.06	1	1.80
December	1	2.89	1	2.77
Average	1	1.38	1	2.12

It may be seen from these figures that the kanduli represented on an average only 1 part as compared to 1.4 parts of biyang-puti in the catch of the baklad in 1931; in 1932 the kanduli decreased further until the ratio was 1 part kanduli to 2.12 parts biyang-puti, whereas about five years ago the kanduli formed the bulk of the catch of the baklad. The replacement of the kanduli in the catch by the biyang-puti is an indication of the rapid depletion of the former in Laguna de Bay.

3. Destruction of immature fish by fishing gear which are suitable for catching fry and very young kanduli.—An examination of the composition of the catch of fishing gear other than baklad and pukot in Laguna de Bay showed the following:

(*a*) *Baklad-siid* or *baklad-guilid* (small, shallow fish corrals) catch at all times almost 100 per cent undersized kanduli, largely because this type of corral is set in shallow water where young kanduli feed.

(*b*) *Sakag*, or scissors nets, catch only very young kanduli, biyang-puti, bañgayñgay (*Ophiocara aporos* Bleeker), and dalag fry.

(*c*) *Seket* (modified type of sakag, or scissors net) is the gear by far most destructive to immature kanduli, biyang-puti, bañgayñgay, and other fishes. These young fish are frequently found schooling in shallow waters where this gear is used. They are driven into the net by dragging along the bottom of the lake an iron chain feathered with banana or buripalm leaves.

(*d*) *Pukot-dalag* also catch kanduli, almost 100 per cent of which are immature.

REMEDIAL AND REGULATORY MEASURES

The obvious depletion of the kanduli in Laguna de Bay changes the problem of its conservation into one of rehabilitation. The following regulative and remedial measures are recommended:

1. The absolute prohibition of the use of pukot-laot or pukot-alañgan (drag seines used in deep water) in Laguna de Bay for at least five years. Five years of study at the College of Agriculture limnological station indicate that the kanduli becomes sexually mature in the second or third year after hatching, and reaches a fair marketable size at the age of about five years.

However, there is a serious objection to the foregoing measure, and that is the question of the pukot to be laid off. Certainly, such a restriction would cause the pukot owners great financial loss. In view of this objection, the following substitute measure is proposed: Every owner or operator of pukot in Laguna de

Bay should be required to register each pukot (drag seine) in the municipality where the owner or operator resides and to pay a yearly license fee for each pukot registered. The following schedule of fees is recommended:

(*a*) For a first-class pukot, 100 pesos a year. A first-class pukot is a drag seine, locally known as pukot-laot or pukot-alañgan, and is operated by at least thirty persons and pulled by a motor boat.

(*b*) For a second-class pukot, 75 pesos a year. A second-class pukot is a drag seine operated by at least thirty persons but propelled solely by man power.

(*c*) For a third-class pukot, 50 pesos a year. A third-class pukot is a drag seine manned and operated by at least fifteen persons, but not more than twenty.

(*d*) For a fourth-class pukot, 25 pesos a year. A fourth-class pukot is a drag seine manned and operated by at least five persons, but not more than ten.

(*e*) For a fifth-class pukot, 10 pesos a year. A fifth-class pukot is a drag seine, known in Laguna de Bay as "pukot-dalag," operated and manned by less than five persons.

Registration of newly made or repaired pukot should not be allowed till five years after the pukot has ceased to be useful. In this way it should be possible to give the kanduli fishery a rest of five years with the least loss to the owners and operators of the pukot. This sacrifice would be well compensated for by the conservation of the kanduli fishing industry.

2. At the expiration of the five-year period the further use of the pukot in Laguna de Bay as a gear for catching fish should be restricted in order to allow for the gradual replacement of the marketable size by the young kanduli. Such restriction of the use of the pukot in Laguna de Bay may be accomplished by the enactment of the following regulatory measures:

Enforcement of a closed season against the use of the pukot in Laguna de Bay during the peak of the brooding activity of the kanduli for the following reasons:

(*i*) The greatest destruction of the eggs, embryos, and larvæ of the kanduli by the pukot occurs during the brooding season as the brooders at this time are very irritable and a slight entanglement in the net causes the nurses to expel the broods from their mouths. The peak of brooding activity takes place from April 15 to June 15.

(*ii*) Closed season against the use of the pukot during the period of heavy spawning, which takes place from March to April, will also undoubtedly help to prevent the unnecessary destruction of eggs, embryos, and young kanduli. However, it must be borne in mind that during this time and about a month prior to the commencement of the spawning season, the kanduli are fat and at their best for human consumption. For a month after the brooding season the kanduli are lean and not commendable for the table.

3. Discouraging the catching of the young, immature kanduli and taking them to the markets, by making it illegal to capture, possess, purchase, or offer for sale, young, immature kanduli, locally known as tiguiti (kanduli measuring 16 centimeters or less from the tip of the mouth to the end of the caudal fin, or tail), and making legal only the sale of kanduli measuring 16.5 centimeters or more in length. The enforcement of the foregoing law would be comparatively simple as the market inspectors, members of the municipal police force, and other deputized persons authorized to enforce such laws could easily check the size of the kanduli offered for sale in public places or in the possession of any person.

4. Ruthless methods of catching prawns, snails, and small fishes in Laguna de Bay, which serve as the major food supply of the kanduli, should be regulated in order to prevent their depletion by unnatural and artificial means.

5. The use of the seket should be entirely prohibited in Laguna de Bay. This gear has proved to be very destructive to the immature kanduli which feed along the shallow shores of the lake. The seket is a type of scissors net (sakag in Tagalog) used in conjunction with an iron chain feathered with buri-palm or banana leaves. The iron chain is dragged by two men along the bottom of the shallow waters towards the scissor net which is set at some distance from a point where the dragging of the chain starts.

CONCLUDING REMARKS

It is apparent from the discussion of the foregoing program of conservation and rehabilitation of the apparently depleted kanduli fisheries of Laguna de Bay, that there is a need for discouraging or even prohibiting the use of the pukot (drag seines) in Laguna de Bay, as this gear has been found most destructive to the kanduli fishery, as shown by the following:

1. At the commencement of and during the breeding season the kanduli have the habit of schooling together. Obviously

during this time the kanduli are caught in large numbers in any single haul of the pukot.

2. In the study of the composition of the catch of the pukot it has been shown that this gear withdrew 60.04 ± 0.41 per cent of the total number of kanduli taken from Laguna de Bay in the 1931–1932 period. Likewise, during the same period, the pukot caught 57.90 ± 2.185 per cent of immature kanduli. This percentage of immature kanduli does not include the countless eggs, embryos, and larvæ that are destroyed by the pukot during the spawning and brooding seasons.

3. Laguna de Bay is a shallow sheet of water, its depth in the open ranging from 8 to 16 feet. The efficiency of the drag seine in catching large numbers of kanduli is very great at this depth, and Laguna de Bay is too shallow a body of water to withstand the severe effects of efficient fishing gear such as the pukot.

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ORIGIN OF EMBRYOS IN THE STRAWBERRY MANGO[1]

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THREE PLATES

February 22, 1913, seedlings of several varieties of Indian mangos were received by the College of Agriculture from Moanalua Agricultural Experiment Station, Oahu, Hawaii, through the courtesy of Mr. J. E. Higgins,[2] now director of the Panama Canal Zone Experiment Gardens but at that time on the Hawaii Experiment Station staff. One of these varieties was the strawberry mango, which was planted in the college orchard near the Protestant Chapel, and has been bearing fruit (Plate 2) for the last few years. A full description of the fruit is given by Higgins(4) and Wester.(13)

April 11 and May 11, 1933, one hundred fruits were collected and their seeds germinated in seed boxes in the department of plant physiology, College of Agriculture; ninety-nine germinated. Twenty-four seeds gave rise to one plant each; twenty-five, two plants each; thirty-eight, three plants each; and twelve, five plants each (Plate 3, fig. 1).

Because of the development of more than one plant from a single seed of this strawberry mango, a feature very uncommon in the Indian mangos, two questions have arisen. Do these embryos develop vegetatively or apogamously, and from what tissue or tissues of the young seed are they formed? Does the megagamete, or egg, whether fertilized or not, take an active part in the formation of any or all of the embryos within a single seed? The answers to these queries are, in the writer's opinion, necessary adjuncts in the hybridization of the mango.

Material was collected during April, 1933, when the blooming period was at its height, with the hope of finding out the probable origin of these multiple embryos in the mature seeds of this particular Indian strawberry mango imported from Hawaii. Collection of early stages of the development of the flower were not then possible, so only mature flowers and fruits at different stages of development were gathered. These were killed and fixed in the laboratory with the use of formalin-acetic-alcohol (70 per cent) prepared according to the formula given by Chamberlain.(2) The material was dehydrated and infiltrated as usual, embedded in paraffin, and cut into sections 6 to 10 microns thick. The sections were stained in Heidenhain's iron-alum-hæmatoxylin with orange G dissolved in clove oil as a counterstain.

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[1] College of Agriculture Experiment Station contribution, No. 976.

[2] Thanks are here expressed to Mr. J. E. Higgins, director of the Panama Canal Zone Experiment Gardens, for identifying this mango.

OBSERVATIONS

The youngest stage obtained in the development of the megagametophyte is the binucleate stage (Plate 1, fig. 1) in which the embryo sac has considerably elongated and the two daughter nuclei lie at both ends; between the two nuclei is the large central vacuole. Stages earlier than this were not available, but the writer is of the opinion that they are perhaps identical with that reported by Juliano and Cuevas(5) for the pico mango from the Philippines. The two nuclei described above undergo the usual divisions, and a normal seven-celled megagametophyte already described for the pico mango(5) is formed. According to Maheswari(6) Dudgeon in 1929 found degenerations in the carpels of mangos in India, beginning as early as the megaspore mother-cell stage. As a result of these degenerations only a few of the flowers are physiologically bisporangiate, and these are towards the apex of the inflorescence. Towards the close of the flowering season the flowers are often completely sterile and fall off in large numbers. As a consequence, the greater percentage of the flowers formed never produce fruits at all as is true of the pico(5) and the carabao(11) mangos in the Philippines.

That pollination is necessary for the production of mango fruits is well shown by the results obtained by Popenoe(8) working in Florida and Juliano and Cuevas(5) in the Philippines.

After fertilization the synergids and the antipodal cells degenerate. The primary endosperm nucleus (Plate 1, fig. 2) then enlarges and becomes rhomboid. Its nucleolus is large and conspicuous and takes the stains very heavily. The zygote, which contains a small oblong to oblong-ovate nucleus embedded in a thin peripheral cytoplasm, is found just below the primary endosperm nucleus. At its center is a large vacuole.

From this stage on the embryo sac experiences rapid enlargement and elongation. Concomitant with this enlargement of the embryo sac, the nucellar cells abutting it become conspicuously engorged with dense cytoplasm, especially at the micropylar end of the nucellus. Absorption and destruction of the nucellus by the embryo sac is so extensive that at times the latter nearly comes in contact with the testa at the micropylar end of the young seed (Plate 3, fig. 2). This is epecially true at the time the embryos are quite visible in the embryo sac.

Endosperm.—The primary endosperm nucleus may remain in the vicinity of the zygote (Plate 1, figs. 2 and 3) and usually becomes enlarged before entering active divisions. The endosperm is of the nuclear type, and its development follows very closely that which has been reported for the pico mango.(5) The nucellus is totally destroyed by the endosperm, and the endosperm is completely absorbed by the embryos in the mature seed.

Zygote.—The zygote remains undivided for a long time after fertilization, and may never show any signs of activity at all, even after endosperm formation has long been under way. Usually, long before the primary endosperm nucleus initiates its first division, the zygote (Plate 1, fig. 3) starts to degenerate. Its nucleus becomes densely stained, and loses its regular outline. It actually sinks into a structureless mass preparatory to complete degeneration. In some cases the zygote may persist a little longer (Plate 1, fig. 4), although showing distinct signs of degeneration; at this time there can be found numerous free endosperm nuclei in the embryo sac. In a very few cases, even after the seed has attained a length of about 2.75 millimeters and numerous free endosperm nuclei are present in the embryo sac, the zygote may remain undivided (Plate 1, fig. 5). Its cytoplasm is greatly plasmolyzed, and its nucleus undergoes degeneration. At any rate, even if the zygote does not degenerate soon after fertilization and may stay in the embryo sac unchanged in this strawberry mango, it does completely disappear and does not take any active part in the formation of any of the embryos in the mature seed.

In the polyembryonic No. 11 mango from Florida, Belling(1) also found that the megagamete, whether fertilized or not, never gave rise to any of the embryos in the sac. On the contrary, as is true in the pico mango from the Philippines,(5) the zygote which may hibernate for a long while in the embryo sac, does persist and is easily distinguishable. This zygote divides transversely later and gives rise to an elongated basal cell and a rounded apical cell. In the pico mango, therefore, one of the embryos in the mature seed is normally the result of fertilization. Sometimes the zygote in the pico mango may actually degenerate, so that the resulting embryos in the seed are all apogamic in nature, an instance similar to that being reported for the strawberry mango in this paper. In other words, the strawberry mango cannot very well be utilized as pistillate material for hybridization work, because of the fact that its zygote degenerates and never gives rise to an embryo in the mature seed. It can, however, be used perhaps as a pollinizer. The pico mango, however, may be utilized as a pistillate plant to some extent. Nothing can be said as to the suitability for hybridization purposes of the other Philippine varieties of mango until a thorough study of the exact nature and behaviour of their zygotes and their extra embryos is made.

Adventive embryos.—The enlargement of the embryo sac and destruction of the nucellar tissue after fertilization occur simultaneously. As the destruction of the nucellar tissue proceeds, the nucellar cells directly abutting the embryo sac become filled with dense cytoplasm. Certain among them, especially those around the micropylar region of the nucellus, became greatly enlarged (Plate 1, fig. 6) and these enlarging cells are easily distinguished from their surrounding cells by their specially larger nuclei, denser cytoplasm, and also by their response and behavior to stains. These differentiating cells are usually deep-seated at first, and as their surrounding nucellar cells are absorbed and destroyed by the embryo sac, they begin to enlarge. They usually round off first, and then start to divide forming adventive proembryos (Plate 3, fig. 2). Numerous nucellar cells may show signs of greater development at nearly the same time, but only a few of them actually divide and form adventive embryos. In fact, a majority of these differentiating nucellar cells never divide at all and eventually degenerate (Plate 1, fig. 6). Adventive embryos of nucellar origin are also reported in *Citrus trifoliata,*(8) *Funkia, Nothoscordon, Citrus aurantium, Euonymus, Coelebogyne, Clusia,* and *Opuntia,*(3) and in pico mango(5) and No. 11 Florida mango.(1)

Examination of hundreds of slides of sections of young seeds reveals the fact that numerous adventive embryos may start to grow simultaneously (Plate 3, fig. 2), but the maximum number which usually attain maturity and actually germinate from a seed is five (Plate 3, fig. 1). Belling(1) observed as many as half a dozen adventive embryos developing in the nucellus of the polyembryonic No. 11 mango in Florida, while three to four are commonest in the pico mango.(5) These adventive embryos project themselves into the embryo sac and exhibit irregularity in shape. Because of the irregularity in the mode of divisions of the cells of the proembryos (Plate 1, figs. 8 and 9), the writer was not able to follow very closely their development in their early stage. These adventive embryos are devoid of well-developed suspensors, and display also a great variety of sizes.

Adventive embryos in the strawberry mango are not only formed from those nucellar cells directly abutting the micropyle of the seed, but also from those nucellar cells on the side of the sac opposite the funiculus (Plate 1, fig. 7). Such origin of adventive embryos was also noted in the pico mango.(5)

Wester(12) states that as the Indian mangos fruited in Florida it was noted that the seeds were monoembryonic, and with one exception subsequent observations in Florida and the West Indies have shown this to be a constant character in all Indian mangos that have fruited there. Rolfs(10) observes that polyembryony is only true in Cambodiana, No. 11 Florida, and the Bombay. Amini on the other hand, produced many fruits without seeds in 1914 in Florida, a small number with one embryo each, and two seeds appeared to be polyembryonic. Wester(12) cites Kinman as having concluded that the Indian mangos Amini, Bulbulchasm, Sandersha, and Totapari are adapted for propagation by seeds, because of their variability. Cambodiana is the only exception. Mr. Hartless, of India, in a letter to Wester(12) stated that Indian mangos in his locality were all monoembryonic, thus confirming the conclusion arrived at in Florida. According to Mendiola,(7) Popenoe, and Burns and Prayag stated that, practically speaking, in India where there are probably some five hundred mango varieties, there is no known polyembryonic mango. In other words, polyembryony in Indian mangos is of rare occurrence, while of the six varieties described by Wester(12) from the Philippines, only the dudul mango was reported not polyembryonic.

In a letter from Mr. J. E. Higgins, of Panama Canal Zone, to the writer the statement is made that "none of these varieties [among which is the strawberry] which you mention reproduce themselves with absolute accuracy, and the seedlings of most of them are quite variable." This is to be expected if the mango variety is of the monoembryonic type like most of the Indian mangos. If the embryos are all apogamous, and the zygote, whether fertilized or not, never gives rise to any of the embryos in the mature seed, as is true of this strawberry mango and the No. 11 race of Florida mango, then the seedlings from them ought to receive and reproduce the indisputable stamp of the parent; that is, if no cytological change in the constitution of any of the adventive embryos takes place during their development. Experience shows that seedlings from this strawberry mango usually come true to type in the College of Agriculture.

The writer was informed that in Hawaii the strawberry mango has not gained much popularity compared with the other Indian mango varieties, because of its inferior fruit.[3] This mango is propagated vegetatively rather than by seed, consequently people have very little occasion to observe any action of its seedlings and learn its embryonic habits. It is known that an Indian mango variety, the Mulgoba,(12) which is monoembryonic, has reverted to polyembryony in its first generation when planted in Florida. It is probable that this strawberry mango has behaved in the Philippines in a manner similar to this other Indian mango in Florida.

SUMMARY

An occurrence of polyembryony in the strawberry mango, an Indian mango variety imported from Hawaii to the Philippines in 1912, is herein reported.

The embryo sac is normal and the endosperm is of the nuclear type.

The megagamete or the egg after fertilization, may persist for a long time in the embryo sac, but it ultimately degenerates and does not take any active part in the production of the embryo or embryos in the mature seed.

All of the embryos found in the mature seed arise from the nucellus just surrounding the micropyle and on the side opposite the funiculus. Many nucellar cells may exhibit that characteristic growth, of which many may only degenerate and fail to form adventive embryos. Numerous adventive embryos may

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[3] Information was furnished by Mr. W. T. Pope, senior horticulturist of the Hawaiian Agricultural Experiment Station, Honolulu, Hawaii.

start to grow, but of these only five or less have been actually observed to develop to maturity in the seed.

Because of the fact that all of its embryos are apogamic in nature, this Indian mango cannot be used as suitable pistillate material in hybridization work. It can, perhaps, be used as a pollinizer.

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ILLUSTRATIONS

PLATE 1. STRAWBERRY MANGO

FIG. 1. A binucleate megagametophyte; × 540.
2. Showing the primary endosperm nucleus (*pen*) and the zygote (*zy*); × 540.
3. An older embryo sac showing degeneration of the zygote (*zy*) and enlargement of the primary endosperm nucleus (*pen*); × 1060.
4. A persistent zygote (*zy*) showing degeneration; × 540.
5. A still more persistent zygote (*zy*) on its way to degeneration; note the free endosperm nuclei (*en*); × 540.
6. Portion of the nucellus (*n*) at the region near the micropyle (*m*) showing embryo initials and young adventive embryos (*aem*); note the free endosperm nuclei (*en*); × 540.
7. Portion of the nucellus (*n*) at the side of embryo sac opposite the funiculus showing an adventive embryo (*aem*); × 540.

FIGS. 8 and 9. Young embryos; × 540.

PLATE 2

Mature fruits and a seed of the strawberry mango, reduced to one-third natural size. Photographed May 26, 1933.

PLATE 3

FIG. 1. Five plants arising from a single seed of the strawberry mango, reduced to about one-half natural size.
2. Photomicrograph showing proembryos (dark bodies) arising from the nucellus near the micropyle of the young seed of the strawberry mango; × 93.

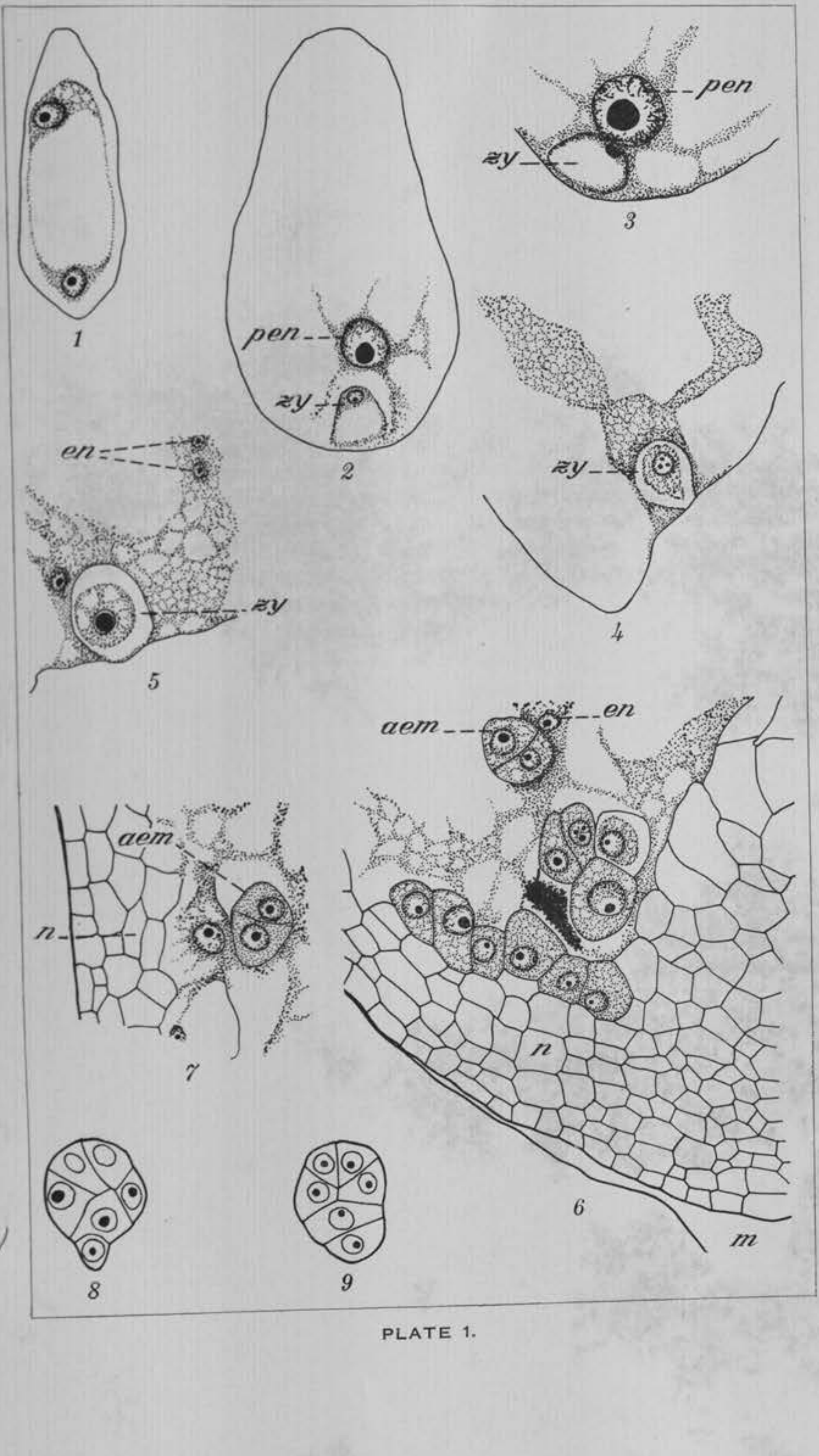

PLATE 1.

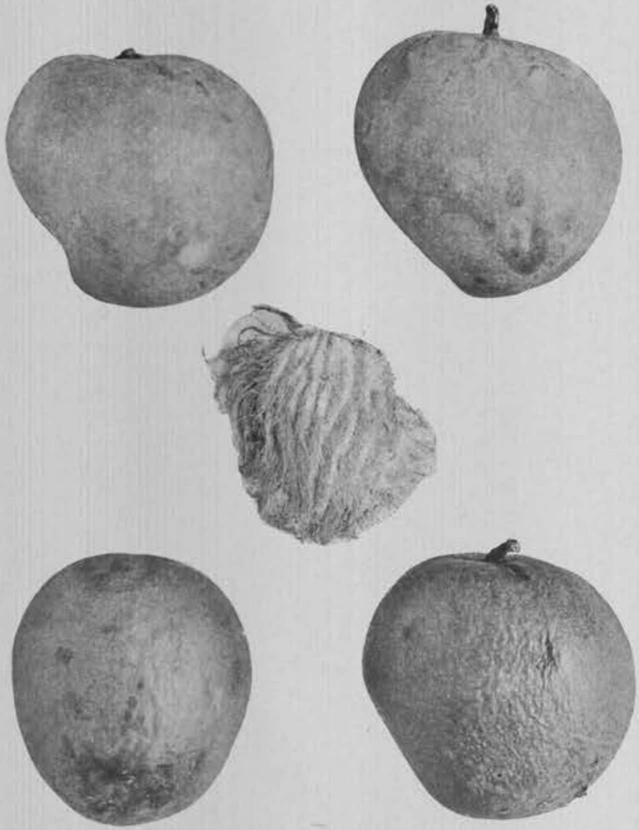

PLATE 2.

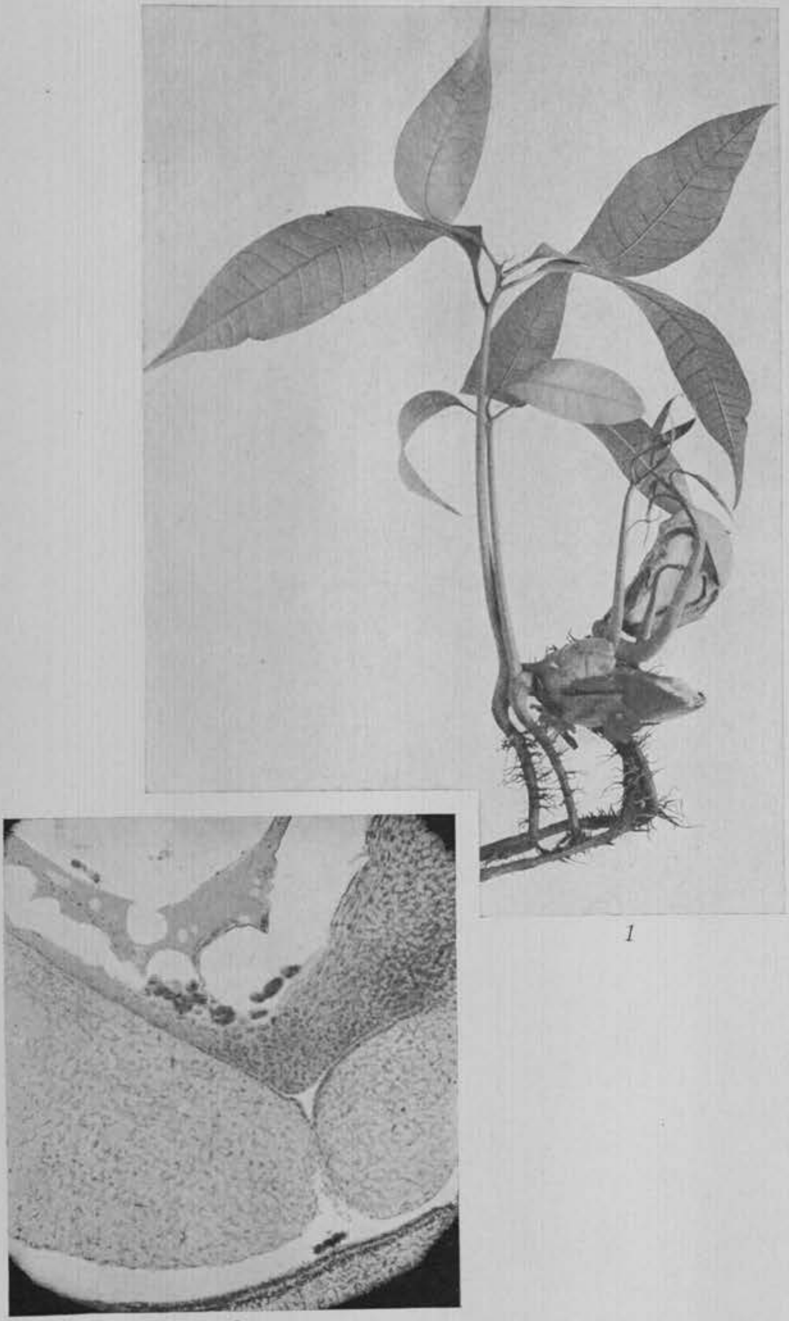

PLATE 3.

ERRATA

VOLUME 53

Page 374, 8th line from bottom, *for* $\frac{\text{35 p.m. minutes}}{\text{a.m. minutes}}$ *read* $\frac{\text{35 p.m. minutes}}{\text{17 a.m. minutes}}$.

Page 375, 5th line from bottom, and page 376, 5th line from top, *for* Table 14 *read* Table 13 in each case.

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